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# Proceedings

of the

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## FOREWORD

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Dr. W. W. Armistead, President of the A.V.M.A. and Dean of the College of Veterinary Medicine, Michigan State University, delivered the major address at the Annual Banquet held during the Convention.

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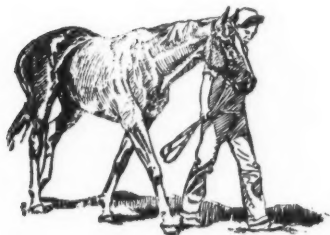
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Papers Presented Before  
THE ASSOCIATION OF EQUINE PRACTITIONERS  
1957 CONVENTION  
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## EXAMINATION OF THE EQUINE HEART

D. K. DETWILER, D.V.M.

### INTRODUCTION

An adequate cardiac examination is obviously of special importance in horses, owing to the nature of their work. This is especially true in race horses and riding horses because of the danger to the rider if his mount should collapse. Although crucial gaps in our knowledge prevent reliable conclusions regarding the incidence of heart disease in horses, it is evident that sudden death is not infrequently attributable to myocarditis (1) or rupture of the heart or great vessels (2). Moreover, a variety of acute infectious diseases of horses are known to produce cardiac lesions which may lead to chronic endocardial or myocardial changes. These pathological processes may be insufficient to cause obvious signs of cardiac impairment except under conditions of strain as during strenuous exercise or during general anesthesia in surgical patients. These considerations emphasize the importance of thorough study of the heart in soundness and preoperative examinations of horses.

### TYPES OF CARDIOVASCULAR DISEASE DESCRIBED IN HORSES

Current knowledge regarding heart disease in horses is fragmentary. It is based largely on postmortem observations, often of the most superficial sort. Sustained, exhaustive investigations of heart disease in horses have not been carried out and there is a particular lack of clinical reports. Owing to this paucity of information, correlation of clinical and necropsy findings is difficult and may be misleading.

Pericarditis occurs most often secondary to pneumonia and/or pleuritis and has been reported in various respiratory infections of horses (2). Distension of the pericardial sac with described in horses having a focus of infection elsewhere in the body. Rubarth (1) described five cases of sudden death in

excess pericardial fluid sometimes occurs with the symptom-complex frequently and historically described as equine influenza (3). As emphasized by Doll *et al.* (4) a variety of viruses and bacteria are involved in the respiratory infections of horses classically described as influenza and they propose discontinuance of this term in diagnosis. At least two different viruses are now known to cause respiratory disease in horses, equine arteritis virus and equine abortion virus (4). Distension of the pericardial sac with excess fluid is described as an occasional finding in viral arteritis of horses (4).

Inflammatory and degenerative lesions of the myocardium occur in horses during the course of various systemic diseases (2, 3, 6, 8). Fatty degeneration and hemorrhage of the myocardium are not infrequent in purpura hemorrhagica of horses. Cardiac lesions are often prominent and extensive in horses which have died from equine infectious anemia. In acute cases hemorrhages and degenerative changes occur. In subacute cases extensive degeneration and induration may be found as well as small areas of infarction. Acute and chronic interstitial myocarditis, often associated with endocarditis, is horses following exercise. A chronic pharyngitis and inflammation of the retropharyngeal lymph glands were found and, on histological examination, changes were seen in the myocardial parenchyma or conduction system. Positive bacterial cultures were found in three cases (*Streptococcus equi* in two and *Streptococcus Type Schutz* in one).

Spontaneous rupture of the heart has been reported relatively often. Undoubtedly, the sudden death and dramatic nature of postmortem findings accounts in part for the frequency with which this accident has been reported. Leonhardt (9) assembled 34 cases: the rupture occurred in the right ventricular wall in 15, right auricle in 7, left auricle in 8, left ventricle in 3, and interventricular septum in 1. The usual cause is underlying myocardial disease such as infarction or myocardial degeneration.

Primary disease of the coronary arteries has not been recognized often and coronary atherosclerosis does not appear to occur as a primary lesion in horses. Myocardial infarcts owing to embolism or thrombosis are occasionally encountered. Bacterial emboli arising from vegetative lesions of bacterial endocarditis account for some of these. Farrelly (10) described four cases of parasitic endarteritis and thrombosis at the origin of the aorta caused by larvae of *Strongylus vulgaris*. In two cases thrombo-embolism of the right auricular branches of the right coronary artery had caused infarction of the right auricle. In a third case, previously reported by Cronin and Leader (10), occlusion of the right coronary artery was caused

by a vegetation located in the aortic bulb. A fourth horse died owing to thrombo-embolism of the common brachiocephalic trunk. Farrelly points out that myocardial infarction is not a common lesion in the equine heart and that, when found, the right auricle is usually involved. The anterior and ventral location of the orifice of the right coronary artery in the aortic bulb would favor entrance of particulate matter, facing as it does the on-coming flow of blood. This is in contrast to the left coronary orifice direction which is almost opposite to that of the right. It is not clear why the auricular branches of the right coronary should be more commonly affected than the other branches. Farrelly described a fifth case in which a healed infarct of the right auricular myocardium was found and suggests that this lesion was probably the result of parasitic thrombosis of the aortic sinuses.

Ossification of the right auricle, especially the auricular appendix, has been reported most frequently in the European literature (2, 5, 6, 7). The accepted view of the pathogenesis of this lesion has been that it is secondary to dilatation of the right auricle with resultant damage to muscle fibers and proliferation of the intermuscular connective tissue, followed by ossification (5). Cohrs and Streich (5) found this lesion in five horses with tuberculosis. Tuberculosis lesions were found in the affected auricular wall in four of these. They concluded that the lesion was an allergic-hyperergic myositis caused by localization of the tuberculous process in the wall of the right auricle in a sensitized animal (5, 6). From the literature it appears that more than half of the horses with this lesion have been found to be tuberculous (5). On the other hand the lesion is also found in horses in which tuberculosis cannot be established, as in Garbers' case (7). Farrelly (10) has suggested that infarction in the auricle resulting from thrombotic lesions in the aorta caused by strongyle larvae may have etiological significance (*vide supra*) since in his three cases the right auricle was involved. The condition has not been described as a clinical entity and is only recognized on necropsy, although in some cases it has been associated with sudden illness and death (5).

Endocarditic lesions are located most often in the left auricle and ventricle (11). The aortic valves are most frequently affected, followed by the mitral, tricusped and pulmonary valves, in that order (11). It is interesting that in 14 serum production horses which developed valvular endocarditis while under immunization with meningococci, all developed purely left heart lesions and the aortic valves were more frequently involved than the mitral (12). The organisms most commonly found in bacterial endocarditis of horses are streptococci, chief-

ly *Streptococcus equi*, although other cocci, *Shigella equirulus* and other organisms associated with navel infections in foals have also been incriminated (8, 11). In horses as well as other species, endocarditis may result as part of an antigen-antibody reaction in previously sensitized animals (13, 14). Chronic inflammatory lesions leading to scarring and induration of the valves (chiefly the aortic and mitral) and mural endocardium are found and when valvular damage is severe enough cause valvular insufficiency or stenosis (rare). Presumably these lesions, which account for some cases of chronic heart disease in horses, represent the healed or terminal stages of tissue reaction to previous bacterial infections or allergic sensitization and response.

Rupture of the aorta occurs most commonly in horses among domestic animals. The usual site of rupture is at the root of the aorta between the aortic valves and the *ligamentum arteriosum*. The aortic wall is relatively thin here (1 mm. thick as contrasted with 5 to 7 mm. at the aortic arch (2)). Local degenerative or inflammatory lesions result in weakening of the vessel wall. Medial necrosis, atheromatous degeneration, arteriosclerosis and parasitic (*strongylus* larvae) arteritis are among the lesions described in these cases. Winterhalter (5) investigated seven cases of spontaneous aortic rupture in horses. The site of rupture in each case was just above the semilunar valves. On examination no visible changes in the arterial wall could be seen macroscopically, but histological sections revealed fine medial necrosis in the region of the rupture. Several cases of dissecting aneurysm of the aorta have been described in this location. Rarely the pulmonary artery is involved and rupture of this vessel has been reported.

In horses with chronic alveolar emphysema, hypertrophy and dilatation of the right heart is a well known post-mortem finding. Although direct measurements of pulmonary blood pressure have not been reported in horses, it is assumed that, as in man, increased pulmonary vascular resistance and resultant pulmonary hypertension cause these cardiac changes. In advanced cases right ventricular dilatation may progress until the tricuspid valve becomes incompetent. A systolic murmur is then audible at the tricuspid area (see Auscultation) and right heart failure with dependent edema, and ascites may supervene, leading to death from congestive heart failure.

## METHODS OF CLINICAL EXAMINATION

### *Palpation*

To palpate the cardiac region on the left side of the thorax place the left hand against the chest wall with the palm in the

cardiac region at the fifth intercostal space and the fingers extending forward under the shoulder musculature toward the third intercostal space and downward toward the sternal margin. Palpation is carried out over the cardiac region on the right thoracic wall, using the right hand.

*Cardiac impulse:* The apex beat of the heart may be palpated in the region of the third to the sixth left intercostal space. It is strongest normally at the fifth intercostal space in the middle of the ventral third of the thorax. On the right side of the thorax the cardiac impulse may be palpable in the region of the third and fourth (rarely fifth) intercostal space in the lower third of the thorax. In heavy muscled horses the impulse may not be palpable here.

*Thrills:* A thrill is a fine vibration which can be felt by the hand. If a heart murmur is sufficiently intense a thrill may be produced. Detection of a thrill establishes the location of the point of greatest intensity of a murmur and indicates heart disease, except in rare cases when palpable thrills are produced by large arteriovenous fistulas (other than patent *ductus arteriosus*) or by an aneurysm.

Forward shift of the caudal limit of the palpable cardiac impulse may occur owing to marked ascites or excessive tympany. Abnormal structures within the thoracic cavity (large abscesses, tumors, etc.) may shift the heart position.

Relative weakness of the cardiac impulse occurs in normal horses with thick thoracic walls, pericarditis with effusion, pleural effusion or any other condition in which a mass is interposed between the heart and the chest wall.

The cardiac impulse is increased in strength under conditions of exercise and excitement, febrile diseases, and in post-block or post-extrasystolic beats.

## PERCUSSION

The presence of gross cardiac enlargement can be demonstrated by delineation of the areas of cardiac dullness through percussion. These areas are situated between the third and sixth intercostal spaces on either side of the chest. To expose the area to percussion the foreleg may be pulled forward as far as possible and comfortable for the subject.

On the left side of the thorax if the left leg is pulled well forward the area of absolute cardiac dullness is found to extend over an area bounded by the edge of the sternum ventrally, the third left intercostal space anteriorly, and an arc passing from a point on the fourth rib about five or six inches above the sternal border through the fifth left intercostal space at

a distance of one to two inches above the sternal border to reach the sternal border in the sixth left intercostal space. If the leg is not pulled forward and the horse is standing with both forelegs equally loaded, the area of cardiac dullness extends over an area bounded anteriorly by the caudal border of the anconeus muscle, ventrally by the sternal border, and dorsally and posteriorly by an arc extending from a point on the caudal border of the anconeus muscle four to five inches above the sternal border to a point at the edge of the sternum in the sixth intercostal space. Thus the area of cardiac dullness on the left side does not extend dorsally in the fourth intercostal space above the middle of the lower third of the thorax.

On the right side of the thorax the area of cardiac dullness is found under the right foreleg extending from the third to the fourth right intercostal space a distance of one to two inches above the sternal border.

These limits of the area of cardiac dullness are only approximations which vary from individual to individual, depending on the shape of the thorax and thickness of the chest wall. When the heart is markedly enlarged or the pericardial sac is markedly enlarged and distended with fluid the area of cardiac dullness may extend dorsally as high as the level of the point of the shoulder and posteriorly to the sixth left intercostal space with corresponding, but lesser, enlargement on the right side.

In the presence of marked pulmonary emphysema the area of cardiac dullness disappears first in the fifth left intercostal space, is lower in the fourth left intercostal space and may disappear entirely on the right side of the thorax.

## AUSCULTATION

The most important part of the physical examination of the heart is auscultation. This must be carefully done and the entire cardiac region on both sides of the chest and under both forelegs examined.

*The Auscultation Areas:* Sounds associated with the various valves of the heart are best heard in different locations and these areas are correspondingly named. The *mitral area* is located at the caudal border of the area of cardiac dullness at the fifth left intercostal space just below the level of a horizontal line drawn halfway between the point of the shoulder and the sternum. The *aortic area* is at the fourth left intercostal space about one inch below the level of the point of the shoulder. The *pulmonic area* is nearby at the third left intercostal space or fourth rib below a line drawn through the middle of the

lower third of the thorax. The *right atrioventricular valve* area is located at the third or fourth right intercostal space in the lower half of the ventral third of the thorax.

*Normal Heart Sounds:* There are four heart sounds, all of which may be heard in some horses. The *first sound* (S1) is associated with closure of the atrioventricular valves and is best heard in the mitral area. Occasionally, this sound is split. The *second sound* (S2) is associated with closure of the semilunar valves and best heard at the aortic and pulmonic areas. This sound is often split. The *third sound* (S3) occurs shortly after the second, is associated with the period of rapid ventricular filling which follows the onset of diastole, and, when audible, is heard best at the mitral area. The *fourth sound* (S4) is associated with auricular contraction, occurs just before the first sound, and, when audible, is heard best at the mitral area.

S4 and S1 are often fused, producing an initial heart sound with two components. S3 and S4 are fainter than S1 and S2 and are not infrequently inaudible. Even when the fourth heart sound is heard at a slow heart rate it will fuse with S1 as the heart rate increases and finally disappear at faster rates. This occurs because with increasing heart rate the interval between auricular and ventricular contraction shortens progressively until, finally, the hemodynamic events producing S4 do not occur, since ventricular contraction takes place first. If the interval between S4 and S1 does not shorten as tachycardia develops and S4 continues to precede S1, the diastolic period may be shortened sufficiently so that S3 and S4 will fuse. This will produce a so-called gallop sound and is sometimes associated with a second sound of decreased intensity. In such cases the predominant or only sounds are S1 and fused S3-S4. Thus two sounds, very close together, are heard.

*Murmurs:* Heart murmurs may or may not signify disease of the heart. They may be transient and due to relatively unimportant functional disturbances, persistent but nevertheless not caused by structural changes in the heart, or persistent and caused by some pathological alteration of cardiac structure and/or function.

A universally accepted terminology for cardiac murmurs which do not have pathological significance, as contrasted with those which do, has not evolved. The term *functional murmur* has been used to designate both physiologic and pathologic murmurs (16). Thus murmurs occurring in otherwise normal hearts after exercise or associated with some alteration in normal function which although not understood, is considered benign are often termed functional murmurs. Likewise, murmurs appearing in some individuals with severe anemia or marked

fever are often called "functional" to distinguish them from those, known as "organic", associated with structural changes of the heart valves, even though they are obviously associated with pathological conditions. The term *organic murmur* refers to bruits caused by valvular deformity and *innocent murmurs* to those not considered of pathological significance.

Likoff and Davie (17) have suggested the term secondary murmur for those associated with anemia, fever, or hyperthyroidism.

White (16) recommended that the terms "functional" and "organic" be abandoned and replaced by "physiologic" and "pathologic", respectively. He recommends further that the "pathologic" designation be subdivided into those of extracardiac causation (e.g., anemia) and those of intracardiac causation (e.g., myocarditis, valvular deformity).

From the foregoing it is clear that certain murmurs do not signify heart disease. Whenever such a murmur is detected in a heart which seems to be normal, the possibility of its having pathological significance must be eliminated insofar as possible.

*Physiologic (functional, innocent) murmurs. Extracardial (cardiopulmonary or cardiorespiratory) murmurs* are caused by friction sounds produced when the heart rubs on adjacent pleural surfaces or by the sound of air being forced into or out of lung tissues by the mechanical action of the beating heart. Those sounds usually vary in intensity with the respiratory cycle and may disappear after exercise or with change in position. They are often transient, usually of low intensity and most often systolic in time.

Niemetz (18) investigated 418 horses and found a systolic, blowing functional murmur over the pulmonary valve in 66.2 per cent. It was best heard on the left thoracic wall, deep under the shoulder musculature in the region of the second and third rib. Reisinger (19) emphasizes that this murmur is intensified by exercise, may vary in intensity in the same individual, and may disappear entirely for a time and then return again. Whether or not a systolic murmur is heard in the pulmonic area in over half of normal horses cannot be confirmed nor refuted by observations in our clinic. It can be said that such murmurs are not uncommon in horses showing no other signs of heart disease and systematic investigation might reveal that, indeed, the incidence is as high as Niemetz's work would suggest.

*Pathologic murmurs not caused by structural changes in the heart valves or by congenital lesions: (Hemic murmurs)* In severe anemia and in febrile states soft, blowing systolic mur-

murs may be heard. Presumably, they are the result of turbulence resulting from lowered blood viscosity in the case of anemia or by increased blood velocity in fever. These murmurs disappear when the underlying condition is corrected.

*Pathologic (organic) murmurs:* The murmurs belonging to this group are associated with acquired structural valvular alterations, various congenital cardiac lesions, and pericarditis.

The valvular lesions produce murmurs which are most intense in their respective auscultation areas. While the correlation between the area where a murmur is most intense and the valve involved is not infallible, there is a fair degree of correspondence. Thus a systolic murmur best heard in the mitral area indicates mitral insufficiency, while in mitral stenosis (rare) late diastolic murmurs as most intense here. Aortic insufficiency is characterized by a diastolic murmur in the aortic area and stenosis by a systolic murmur in the same location. Likewise pulmonic insufficiency is characterized by a diastolic murmur in the pulmonic area and stenosis by a systolic murmur. Right atrioventricular valve insufficiency produces a systolic murmur and stenosis a diastolic murmur in the corresponding auscultation area.

In horses, as in dogs and men, systolic murmurs are most common. The mitral and pulmonic or aortic areas are the sites at which murmurs are most often heard.

The murmurs of certain congenital lesions (*patent ductus arteriosus and pulmonary stenosis*) are characteristic in the dog with respect to location, timing, and quality (20). Similar characteristics probably apply to the murmurs associated with these lesions in the horse but, with the exception of interventricular septal defect, the author has had no opportunity to examine animals with known congenital malformations. In the case of interventricular septal defect the murmur was most intense at the "mitral" area.

A friction sound may be produced when pericardial inflammation is present (*pericardial friction rub*). This murmur is of a grating character and may be heard during both systole and diastole.

When a murmur is intense enough to produce a palable thrill, it is always pathologic. Locating the thrill establishes the area of greatest intensity and the diagnosis of heart disease.

### JUGULAR PULSE

The jugular veins and their contained blood act like an elastic fluid manometer, reflecting pressure changes occurring in the right atrium and in the thoracic cavity. The extent to

which pulsation in these veins is visible depends upon the pressure within the atrium, the amount of tissue overlying the veins, the height of the veins with respect to the level of the right atrium, pulsations transmitted directly from the right atrium, and pulsations transmitted to the right atrium from the ventricles and to the veins themselves from the carotid arteries. Obviously, many of these factors are variable under normal circumstances. Consequently, the interpretation of visible pulsation in the jugular veins must be made with due regard to normal variations.

The normal jugular pulse consists of several waves during each cardiac cycle. When the right atrium contracts a pulse wave is transmitted to the jugular veins. This wave is followed by a second wave due to bulging of the right atrioventricular valve into the right atrium during early ventricular systole and transmission of the pulse from the adjacent carotid artery. As the base of the heart moves downward and the papillary muscles shorten and move the atrioventricular valves downward in systole, a negative wave occurs. This is followed by a positive wave as the atrium and veins fill with blood during ventricular systole. When the atrioventricular valves open the blood flows rapidly into the ventricles and another negative wave is seen. These waves occur in sequence during each cardiac cycle and are further complicated by pressure changes in the thorax and modifications of the wave form resulting from the elasticity of the veins and surrounding tissues and pressure effects from contiguous muscles and organs.

Pulsations and undulations of the jugular veins are usually visible at the thoracic inlet and often for a variable distance along the veins above the level of the heart base. A rhythmic swelling and collapse of the veins may be seen with respiration, the veins filling somewhat during expiration and emptying partially during inspiration. This effect is exaggerated by factors increasing expiratory intrapleural pressure (expiratory dyspnea). If the head is lowered and the veins reach a level below that of the heart, they are seen to fill with blood. In the commonly occurring incomplete heart block with dropped beats (q.v.) the veins may be seen to fill during the doubled diastolic interval.

The auricular wave occurs just before the end of diastole. It can often be identified by visual inspection and its time in the cardiac cycle verified by palpating the apex beat of the heart and the lower end of the jugular furrow simultaneously. It precedes the palpable apex beat by a brief interval. Pressure on the jugular veins at the thoracic inlet blocks this pulse wave, but that owing to the underlying carotid artery pulsation may still be seen in the veins as they distend with blood.

Insufficiency of the tricuspid valve is accompanied by regurgitation of blood which, when the volume is great enough, produces strong pulsation in the jugular veins. The pulse occurs shortly after the apex beat and often seems to approach in strength and character that of an arterial pulse. It is distinguished from the auricular pulse since it occurs later in the cycle (i.e. after the apex beat) and from the transmitted carotid pulsation in that pressure on the vein will abolish it peripherally while that caused by the underlying carotid artery remains.

## ELECTROCARDIOGRAPHY

Brooijmans (21) has recently published an extensive treatise on the normal and abnormal electrocardiogram of horses and cattle which contains numerous examples of electrocardiograms from horses with heart disease and details methods of taking clinical electrocardiograms. Various workers use different leads and methods have not been standardized as yet in equine practice. In the Clinic of the School of Veterinary Medicine, University of Pennsylvania, the following leads are now taken routinely.

*Standard bipolar limb leads:* The three leads which Einthoven originally used in man are employed. They are right foreleg and left foreleg (lead I), right foreleg and left hind leg (lead II), and left foreleg and left hind leg (lead III).

*Augmented unipolar limb leads:* In these leads two of the three limbs used in the standard bipolar limb leads are paired against the third limb. Records are taken from each limb in sequence and are labeled, in accordance with current terminology, aVR, aVL, aVF; R designates right foreleg, L left foreleg and F left hind leg. In each case the limb designated by the letter is connected with one pole of the galvanometer and the remaining two limbs connected together with the opposite pole.

*Chest leads:* An exploring electrode is placed on various locations on the chest and paired with an electrode or group of electrodes at some point distant from the heart. Several chest leads are taken, including one over the left ventricle in the sixth left intercostal space about one inch above a horizontal line through the top of the olecranon and one over the right ventricle in the sixth right intercostal space at the same level. These chest leads are paired against the Wilson Central Terminal in which the two forelegs and left hind leg are joined and connected to one pole of the galvanometer. These leads are labelled respectively CV6L and CV6R. In addition, the chest lead on the left side (6L) is paired with an electrode located on the right side of the neck at the base, on the level of a line drawn

through the most prominent part of the scapular spine. This lead, labelled (I), is taken by placing the right arm lead at the base of the neck, the left arm lead at the cardiac apex (6L) and registering lead I. With the electrodes in this position and the left leg lead on the left hind leg, three bipolar leads are taken in sequence and labelled (I), (II), (III).

The heart rate determined in the electrocardiogram averaged 35 (range, 27-47) per minute in a series of 49 horses (22). Lannek and Rutqvist (23) found the average for cold blooded horses to be 43.6 per minute and 42.6 for warm blooded horses. This disparity in average heart rate may be accounted for by the fact that in the first series the total number of animals was much smaller than that of Lannek and Rutqvist and many of the records were taken without removing the horses from their accustomed stalls.

The durations of the various electrocardiographic intervals are presented in Table I from the data of Lannek and Rutqvist (23).

Table I. Duration of P, PR, QRS, and QT in Horses, Lead II.

		P	PR	QRS	QT	HR/min.
Warm-blooded Horses	Upper probable abnormal limit	0.175	0.429	0.155	0.625	79.3
	Upper normal limit	0.155	0.382	0.140	0.575	70.2
	Mean	0.117	0.286	0.111	0.475	42.6
	Lower normal limit	0.079	0.190	0.082	0.376	24.2
	Lower probable abnormal limit	0.059	0.143	0.068	0.326	19.6
	Upper probable abnormal limit	0.172	0.465	0.139	0.589	96.8
	Upper normal limit	0.154	0.410	0.126	0.567	86.1
Cold-blooded Horses	Mean	0.120	0.298	0.100	0.475	43.6
	Lower normal limit	0.085	0.187	0.074	0.339	27.7
	Lower probable abnormal limit	0.067	0.131	0.061	0.293	22.4

Note: The two values on either side of the mean form statistical boundaries between normal and probably abnormal time intervals. The two outer values are statistical boundaries between probable abnormality and significant abnormality.

Steel (24) on the basis of electrocardiograms, both limb lead and chest leads, taken on 306 horses gives somewhat different values as follows: P wave 0.09 to 0.17 seconds, mean 0.132 second; PR interval 0.22 to 0.66 second, mean 0.325 second; QRS interval 0.09 to 0.18 second, mean 0.122 second; QT interval 0.42 to 0.62 second, mean 0.524 second; mean heart rate 35.9 per minute. The QRS interval averaged from the three standard limb leads on each horse ranged from 0.086 to 0.146 second with a mean of 0.110 second. Although Steel regards a PR interval of 0.40 second or longer as abnormal, this value occurs in horses showing no signs of cardiac disease.

The P wave is usually positive, most often notched, sometimes biphasic in the limb leads. These variations are considered normal by Lannek and Rutqvist (23) and by Van Zijl (25) although Brooijmans (21), on the basis of vector analysis and the well-known variation in P wave form seen in equine electrocardiograms, regards only positive P waves normal in the limb leads. P is negative in lead aVR (Steel (24)). The QRS complex is variable in form and amplitude in the limb leads. Although a prominent R wave is usually present, the amplitudes are frequently small and the entire complex possesses a complicated configuration with slurring and notching of the various waves. The QRS in aVR is usually negative (deep S or QS) and in aVL and aVF usually predominantly positive (Steel (24)).

### ARRHYTHMIAS

The cardiac arrhythmias encountered most commonly in horses are incomplete atrioventricular block with dropped beats, sinus arrhythmia, ventricular premature systoles, auricular fibrillation, and sino-auricular block. Only these will be discussed in the following.

#### *Incomplete atrioventricular (AV) block with dropped beats:*

In this condition conduction of the impulse from the auricles to the ventricles is delayed and intermittently fails altogether. Cardiologists distinguish two types of incomplete AV block with dropped beats. In Type I the auriculoventricular conduction time (PR interval) is increasingly prolonged during succeeding cardiac cycles until it fails entirely and the ventricles miss one or more (rarely) beats. In the post-block complex the PR interval is short once more and the cycle is repeated. This progressive lengthening of the PR interval followed by shortening after the missed ventricular beat is called the Wenckebach phenomenon. In the horse variable PR intervals are characteristic of incomplete AV block with dropped beats and the post-block PR interval is usually shorter than the pre-block interval. However, the post-block PR interval is seldom the

shortest interval found (except when more than one ventricular beat is missed) and the pre-block interval is sometimes not the longest. Thus the variations in PR intervals are not as regular and predictable as in the classical Wenckebach phenomenon described in man and in controlled animal experiments.

In Type II incomplete AV block with dropped beats the progressive lengthening of PR before the missed ventricular beat is absent and the post-block interval is not shorter than the pre-block interval. This type of AV block is rare in horses (21).

Incomplete AV block with dropped beats is common, occurring in up to twenty per cent of horses at rest. Characteristically, it disappears following adequate excitement, exercise, or atropine. The significance of this arrhythmia is controversial. Owing to its frequency in otherwise normal horses it is generally considered a benign, normal variant. On the other hand some workers (21) suggest that the presence of this type of conduction disturbance warrants a guarded prognosis. At present there is no evidence that the conduction system is necessarily diseased in horses with this arrhythmia. Until such evidence is brought forth it seems advisable to consider incomplete heart block with dropped beats a normal variant in horses. Occasionally, it fails to disappear following atropine or either fails to disappear or is induced following exercise. This is an abnormal finding. When blocking vagal action with atropine does not abolish incomplete AV block with dropped beats, damage to the conduction tissue is probable. When it is induced instead of abolished following exercise either damage to the conduction tissue or an abnormal reflex vagal effect is likely. Such reflex effects do not necessarily arise in the heart, but heart disease should be suspected (e.g. myocarditis) until disproven.

*Sinoauricular (SA) block:* In this arrhythmia both the atria and ventricles miss a beat periodically because the impulse does not reach the atria from the sinus node. The sinoauricular node fails from time to time to produce a conducted impulse. It is probable that this is owing to an alteration of the pacemaker activity of the sinus node, rather than an impairment of conductivity in auricular tissue surrounding the node.

SA block is much less frequent than AV block. Like AV block with dropped beats, it is usually abolished by adequate exercise, excitement, or atropine and thus appears to represent a benign vagal effect.

*Sinus arrhythmia:* The term sinus arrhythmia designates a periodic variation in the frequency of discharge of the sinoauricular node. There are two chief forms: *respiratory*, in which the variations are related to the breathing cycle and *non-*

*respiratory*, in which this relationship cannot be demonstrated. Both types occur in horses. Sinus arrhythmia is common in horses and is always present in incomplete heart block with dropped beats. Sinus arrhythmia, in itself, is not an abnormal phenomenon, although it may be exaggerated or induced by morbid processes which affect vagal or sympathetic tone.

*Ventricular premature systoles:* Ventricular premature systoles are cardiac contractions which arise earlier than expected in an ectopic pacemaker located in the ventricles. Although the diagnosis of premature beats often may be made by auscultation of the heart or palpation of the pulse, determining the location of the pacemaker within the ventricles depends on the electrocardiogram. On the basis of present limited information, it seems advisable to regard ventricular premature systoles as evidence of pathological involvement of the heart, especially when induced following exercise. If the extrasystoles are frequent and if they arise from two or more foci within the heart (on the basis of electrocardiographic configuration) the degree of cardiac damage may be considered greater than when they are infrequent and arise from a single focus.

*Auricular fibrillation:* In auricular fibrillation the auricles fail to contract in a coordinated fashion; instead they remain in diastole, while irregular contractions of small groups of fibers occur throughout the musculature in a completely chaotic pattern. This auricular activity is reflected in the electrocardiogram as small, irregular waves occurring at a frequency of from 300 to 500 or more per minute (26). The ventricles respond irregularly to these impulses, usually at an increased frequency although the rate may be within the normal range or even slower than normal. Owing to the irregularity in ventricular rhythm, the intensity of heart sounds are characteristically variable.

The clinical diagnosis of auricular fibrillation, without the aid of an electrocardiogram, may be based on the following criteria (27):

- 1) An absolutely irregular heart rhythm. Typically, a series of rapid beats is succeeded by pauses of varying duration which, in some cases, may last up to ten or fifteen seconds.
- 2) Elevated heart rate. Although the heart rate is usually increased, it may be normal or slow.
- 3) The pulse rate may be less than the auscultatory heart rate when the two are counted simultaneously (pulse deficit). This occurs when the ventricular rhythm is irregular with the result that some contrac-

tions follow the preceding systole so closely that sufficient blood has not entered the left ventricle to produce a palpable pulse. Pulse deficit diminishes or is absent at slow ventricular rates.

4) Occasionally, the second heart sound is absent when insufficient blood is in the ventricles at the time of systole to open the aortic and pulmonary valves.

5) The auricular or fourth heart sound is never heard. This is a useful characteristic in distinguishing auricular fibrillation with slow ventricular rate and fairly regular rhythm from incomplete heart block with dropped beats. In the latter arrhythmia detection of the fourth heart sound during prolonged diastolic intervals confirms the diagnosis.

6) The heart sounds and murmurs vary in intensity owing to the arrhythmia.

7) The pulse is variable in force.

It has been found that auricular fibrillation can be induced in horses anesthetized with chloral hydrate by the intravenous injection of epinephrine hydrochloride (26). In limited trials the arrhythmia was induced in three out of five horses using the following doses: chloral hydrate, 0.12 Gm. per kilogram of body weight and epinephrine hydrochloride, 0.025 mg. per kilogram of body weight. These experiments were carried out in part because in one of our cases (26) the arrhythmia appeared in a horse with valvular heart disease following surgery, in which chloral hydrate was employed for surgical narcosis and a local anesthetic containing epinephrine had been injected. It is possible that, in a susceptible individual, the epinephrine produced accompanying struggling and recovery from chloral hydrate anesthesia might result in the production of auricular fibrillation.

Auricular fibrillation may occur under various circumstances and its clinical significance depends on the severity of the underlying heart condition. It can develop transiently, in apparently otherwise normal hearts, then disappear completely (26). Usually it is associated with serious heart disease and should be considered a grave prognostic sign. However, several cases have been reported in which horses with auricular fibrillation continued to perform useful work.

With respect to treatment, one cannot disagree with Brooijmans' (21) point of view that, in principle, an attempt should always be made to abolish the arrhythmia. Even

in horses which can work despite the presence of the arrhythmia, their stamina is always reduced and, in the case of riding horses, they are dangerous to use because of the possibility of syncope. Experience with treatment is limited, but, the arrhythmia was abolished in the four cases we have treated with quinidine sulfate. In the first case the daily oral dose of quinidine sulfate was gradually increased from 12 grams to 40 grams, given in divided doses. Then hourly doses of ten grams each were given for a total of 90 grams and during the night following this course of quinidine, the rhythm returned to normal. The second horse was given the same 90 gram course of treatment and likewise converted to sinus rhythm during the night. The nasal mucosa became engorged during therapy and epistaxis occurred in the first animal. Recently, two additional cases became available for treatment. It was decided to give three doses of 20 grams each at three hour intervals. Three hours after the second dose normal rhythm appeared, marked swelling of the nasal mucosa developed, and it was feared a tracheotomy might be necessary. However, the nasal passages remained patent and the respiratory difficulty diminished during the next twelve hours. This horse was given a daily maintenance dose of quinidine sulfate of ten grams for five days and discharged three days later from the hospital. A report from the owner four months later indicated the rhythm had remained normal. A more cautious dose schedule was planned for the next animal treated. On the first day of treatment a test dose of 5 grams was administered. This did not produce any toxic signs and the nasal mucosa remained normal in appearance. For the next two days a total of 20 grams was given in two 10 gram doses several hours apart. Auricular flutter appeared in the evening on both days, but auricular fibrillation was present the following morning in each instance. For the next three days four 10 gram doses were given daily at four to five hour intervals. Auricular fibrillation continued until the morning of the fifth day when auricular flutter appeared, indicating persistence of quinidine action. Two additional 10 gram doses were administered and by evening auricular tachycardia with 2:1 AV block had developed. At the same time there was swelling of the nasal mucosa and some breathing difficulty. The following morning the pulse was regular and normal sinus rhythm had been re-established. Daily doses of 20 grams of quinidine (two divided doses) were continued for the next three days and 10 gram daily doses for the following three days. The horse was discharged after five days without medication. A report from the owner four months later indicated the arrhythmia had not returned.

On the basis of these trials the following dose schedule is suggested:

1st day	5 grams test dose
2nd day	10 grams 2 x daily
3rd day	10 grams 3 x daily
4th day	10 grams 3 x daily
5th day	10 grams 4 x daily
6th day	10 grams 4 x daily
7th day	10 grams 5 x daily
8th day	10 grams 5 x daily
9th day	15 grams 4 x daily
10th day	15 grams 4 x daily
11th day	20 grams 4 x daily
12th day	20 grams 4 x daily
13th day	22 grams 4 x daily

These doses should be given at four hour intervals if possible. Electrocardiograms should be taken morning and night and the nasal mucosa examined for swelling which might interfere with breathing. This recommended dose schedule should only be considered provisional and dosage should be adjusted in accordance with the response of the patient.

After conversion to normal rhythm maintenance doses may be unnecessary. If auricular fibrillation recurs, the successful dose should be employed again and gradually reduced during a period of seven to ten days. Recurrence after this indicates that the condition cannot remain abolished without daily medication and this would be undesirable under most circumstances.

#### EXERCISE TEST AND ITS SIGNIFICANCE

As could be anticipated, it has been found that exercise may bring out electrocardiographic abnormalities in animals with diseased hearts and in conditions in which anoxemia develops. Records showing such changes have been published by Sporri and Leemann (28) and especially by Brooijmans (21).

The term coronary insufficiency has been used in cardiology to designate any condition causing inadequacy of the coronary circulation to meet metabolic needs of the myocardium. Essentially it signifies myocardial hypoxia from any cause. It does not imply primary coronary artery disease with narrowing or occlusion, although this is one cause of coronary insufficiency. Lepeschkin (29) devotes a chapter to this condition as seen in man and Brooijmans (21) has employed the concept embodied in the term to designate the complex of possible conditions which underlie specific abnormal changes that may occur in the electrocardiogram of the horse following exer-

ise. Both these works should be consulted for a more detailed explanation of the various conditions which may cause coronary insufficiency.

Brooijmans (21) has compared changes in the electrocardiogram of normal horses after exercise, based on the data of Landgren and Rutqvist (30), with changes noted in abnormal horses and, on the basis of a limited number of cases, concluded that the following changes in the electrocardiogram immediately after exercise are characteristic of coronary insufficiency:

1. Elevation of the RS-T segment in Leads II, III, and aVF; depression of RS-T in lead aVR.
2. The occurrence of arrhythmias of all types.
3. The occurrence of conduction disturbances.
4. Elevation or depression of T waves beyond normal values.

It is important to emphasize that these alterations can be caused by a variety of conditions which produce myocardial anoxia and do not, necessarily, indicate primary disease of the heart. For example Brooijmans found pronounced changes in a horse with paralysis of the recurrent laryngeal nerves (roaring) which became anoxic when exercised. Following surgical correction and establishment of a free airway the signs of coronary insufficiency following exercise were absent.

Brooijmans (21) has established the following method for an exercise test:

- 1) Using a direct writing electrocardiograph control limb lead and precordial lead electrocardiograms are taken.
- 2) The animal is exercised. Riding horses are made to trot or gallop or trot on a lunge line and draft horses are put to pulling a light harrow. The amount of exercise is determined by the previous training history and the ability of the subject to perform work.
- 3) As soon as possible after exercise electrodes are attached and continuous records of the limb leads are taken for two minutes. Short records of all limb leads are taken at 3, 4, 5, 10, 15, 20, and 30 minutes after cessation of exercise.

In our clinic post-exercise electrocardiograms are taken whenever possible, using essentially the method outlined in the foregoing. To facilitate immediate recording after exercise the points where electrodes are to be placed are well prepared beforehand with electrode paste. The electrodes are pro-

vided with handles and a man for each electrode stands ready for immediate application of the electrodes as soon as the horse is stopped. In this way little time is lost. Precordial leads as well as limb leads are employed. At the track or on farms, riding horses are trotted right up to the recording area and the electrodes applied as the rider dismounts.

Before applying the exercise test, the clinician must familiarize himself with the changes in the electrocardiogram of normal horses after exercise. Parameters for these changes are given in the paper by Landgren and Rutqvist (30) already cited. With increased heart rate there is shortening of all the electrocardiographic intervals (e.g. PR, QRS, QT, etc.) and characteristic T-wave changes (e.g. elevation of the T-wave in lead (I) or CV6L).

### CIRCULATION TIME TEST

Circulation time tests are employed to determine the speed of blood flow. They do not measure the actual blood velocity, which varies with the diameter of the blood vessels, but rather the interval between injection of a substance and its arrival at some distant part of the body. It represents the approximate reciprocal of the average blood velocity between the two points and is related to the cardiac output and the volume of circulating blood. The chief value of such tests is to confirm the clinical diagnoses of congestive heart failure.

Louf and Bouchard (31, 32) have obtained data on the circulation time in horses employing a solution of lobeline (lobeline "Ingelheim" of the firm C. H. Boehringer) containing 10 milligrams per ml. The dose used was 14 milligrams per 100 kilograms of body weight, injected within  $\frac{1}{2}$  second into the jugular vein. This dose produces a transitory hyperpnea which starts abruptly, persists  $\frac{1}{2}$  to 2 minutes (respiratory rate 26 to 30 per minute) and then disappears. Secondary reactions include a brief pre-reaction apnea, loud breathing (which is exaggerated in animals with emphysema and pulmonary sclerosis), and a cuplike depression at the thoracic inlet at the onset of the first reflex inspiration.

With this method in horses showing no sign of disease the circulation time, measured from the time of injection until the first recognizable response, was from 15 to 35 seconds (average 25 seconds). In another series of horses in which auscultation revealed disturbances of rhythm or murmurs without signs of functional disability, the circulation time varied between  $17\frac{3}{5}$  to  $29\frac{2}{5}$  seconds with an average of  $23\frac{3}{5}$  seconds. These animals

were classified as compensated cardiac cases. In horses with heart disease and cardiac decompensation the circulation time exceeded 35 seconds (range 36 to 51 seconds).

The relationship between circulation time (CT), cardiac output (CO), and blood volume (BV) can be expressed as follows:

$$CT = K \frac{BV}{CO}$$

in which K represents a constant that may vary in different individuals or in the same individual under changing conditions. It is clear from this relationship that if blood volume and cardiac output increase to the same extent the circulation time will not change. If the blood volume increases and cardiac output decreases, as occurs in advanced congestive heart failure, the circulation time will be increased. An increase in the cardiac output with no change or a decrease in blood volume will result in shortening of the circulation time. Thus reduced circulation time is to be expected during and following exercise and, in man, is known to occur in such pathological conditions as hyperthyroidism, anemia, and febrile states.

The practical usefulness of this circulation time test in horses remains to be established. The respiratory stimulant effect of lobeline is attributed to its action on the carotid body. In experimental animals this effect is accompanied by a rise in blood pressure, and cardiac slowing and, in man, a sense of strangulation and intestinal contraction (33). The pharmacological actions in horses have not been carefully studied and, other than the report of Louf and Bouchard (31, 32), little is known regarding undesirable reactions which might occur in horses with cardiac disease or other diseases. Certainly, a less potent and less potentially harmful drug would be more desirable for clinical use. In man subjective sensations of the patient can be used to detect the arrival of test substances such as sodium dehydrocholate (tongue, bitter taste), saccharin (tongue, sweet taste), and calcium gluconate or magnesium sulfate (hot sensation in tongue and pharynx). Radioactive sodium ( $Na^{24}$ ), which may be detected by a Gieger-Mueller counter at any distant point, fluorescein or riboflavin which may be visualized as they arrive in the mucous membrane of the lips or tongue viewed under ultraviolet light, inhaled gases (e.g. carbon dioxide which produces respiratory stimulation and nitrogen or helium which reduce arterial oxygen saturation) and dyes (e.g. T-1824) have all been employed. These objective methods avoid undesirable side-reactions but have the disadvantage of requiring costly apparatus to detect the end point.

Thus, while a circulation time test presumably would be of value one cannot be recommended at this time for general use.

## DIAGNOSIS OF HEART DISEASE

The common signs of heart disease in horses include: 1) systolic murmur, 2) dyspnea, 3) cough, 4) edema, and 5) loss of stamina. Since all of these may be present in the absence of primary disease of the heart, they cannot be relied upon in diagnosis.

There are certain signs which, while not always present in heart disease, can be considered reliable evidence if one or more is present. These reliable signs are:

- i Arrhythmias
  1. Auricular fibrillation (or flutter)
  2. Complete atrioventricular block
- ii Murmurs
  3. Pericardial friction rub
  4. Thrills
- iii Dynamic
  5. Generalized venous engorgement

Auricular fibrillation seldom if ever occurs in the absence of primary heart disease (26) and complete heart block always indicates cardiac damage. A pericardial friction rub occurs when the pericardium and epicardium are involved in a pathological process which produces roughening of these surfaces. When a heart murmur is intense enough to produce a thrill either a congenital or acquired lesion is present. Generalized venous engorgement is likely to be associated with heart disease. The jugular veins would be distended and ascites or pleural effusion are likely to be present. Ascites is often difficult to detect in horses except by withdrawing fluid with a hypodermic needle.

Signs of heart disease which are of controversial significance or of questionable specificity include: 1) ventricular extrasystoles, 2) all arrhythmias appearing after exercise, 3) RS-T segment shifts, 4) wandering pacemaker, 5) diastolic murmur, 6) persistence of prolonged PR interval after exercise and 7) enlargement of the area of cardiac dullness.

Ventricular extrasystoles are of almost universal occurrence in man and in most instances are not associated with detectable heart disease. They have been reported in otherwise normal horses (34) although the presence of a myocardial lesion could not be ruled out. Further information is needed to assess the clinical significance of ventricular extrasystoles. Until this is forthcoming it seems advisable to base the diagnosis of heart disease on other criteria. However, if ventricular

ectopic beats are frequent, arise from multiple foci and are increased following exercise, it is most likely that myocardial disease is present.

Arrhythmias appearing after exercise are known to occur in horses with diseased hearts and have never been reported in healthy animals. Until evidence to the contrary is adduced, it seems advisable to consider their appearance as a sign of heart disease.

RS-T segment shifts at rest or after moderate exercise occur in myocarditis and/or anoxia. Obviously, the heart may not be diseased if the anoxia is caused by some abnormality in the respiratory tract. In the absence of this heart disease should be suspected.

Wandering pacemaker is defined as a continuing change in the location of the pacemaker in the auricles and is characterized by changes in the form and duration of the P wave and sometimes by variations in the PR interval. Brooijmans (21) considers this a pathological finding in horses. Since this phenomenon has been observed 16 times in a series of 48 horses considered clinically normal, it is difficult to agree with this interpretation (22). Moreover, a similar phenomenon is not rare in dogs showing no evidence of heart disease. For these reasons this should be considered a normal variant at present.

Diastolic murmurs are occasionally heard in horses which have no other signs of cardiac disease. They occur most commonly at the pulmonic and aortic areas. There is insufficient evidence to draw conclusions regarding the clinical significance of such murmurs. Other signs of heart disease should be carefully searched for when murmurs are found but, if none are found, the diagnosis of heart disease cannot be supported by the presence of a diastolic murmur alone.

Persistence of prolonged PR interval after exercise suggests the presence of a conduction disturbance which may have as its basis pathological changes in the conduction system. This phenomenon has occurred in some horses in our clinic but it has not been possible to follow any of these cases to learn whether or not it disappeared or other evidence of heart disease developed. For the present, it is considered an abnormal finding of probable pathological significance.

Enlargement of the area of cardiac dullness may be caused by any mass, including the heart, which displaces normal lung tissue in this area. Obviously, other things than an enlarged heart may be responsible. Thus, while the only way to diagnose cardiac enlargement in the horse is through percussion and palpation, this can only be considered an unreliable sign of heart disease.

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#### FOOTNOTES

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## PRELIMINARY STUDIES IN EQUINE INFECTIOUS ANEMIA

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The problem of infectious anemia in horses is not a new one. It was first reported in France by Lignee in 1843. Watson in 1896 was the first to describe this disease in the United States. Carre and Vallee demonstrated it to be caused by a filtrable virus in 1904 in France. In the United States during the past five decades the disease has been reported authentically in 34 of the 48 states. The last major outbreak in recent years was in 1947 in New Hampshire. In this outbreak eighty thoroughbreds were lost. It appears, according to the literature, that the disease exists in all areas of the globe with the exception of Great Britain and the Iberian peninsula.

An extensive study was conducted at the school of Veterinary Medicine, University of Pennsylvania, under the sponsorship of the Grayson Foundation from 1948-1953, under the direction of Dr. Kelser. Dr. M. N. Dreguss and Dr. Louise S. Lombard prepared a monograph that was published in 1954. This publication has been a valued guide for the investigations conducted by our group and we are sincerely grateful. It not only describes their own studies but also contributes an excellent and thorough review of prior investigations by many scientists. It is a real contribution to veterinary medicine.

In Florida, the disease has been seen occasionally in the lowlands throughout the state. It has been known that there are enzootic areas with seasonal outbreaks. A rather severe outbreak in 1954 precipitated the current study at the University of Miami School of Medicine. The diagnosis in this outbreak was confirmed by animal test and due to the many disadvantages of this method of diagnosis, a search for a simple laboratory procedure was renewed. The financial support for this project, to date, has been contributed by Mr. James Donn of the Gulfstream Racecourse.

Our study was begun with the use of clinically positive sera from a group of thoroughbreds, on a single farm, all of whom had acquired the infection naturally. As the study progressed, an attempt was made to experimentally infect a horse

with the virus from the New Hampshire epizootic. A lyophilized specimen was obtained from the U. S. Bureau of Animal Industry, reconstituted in 10 ml. of sterile water and intravenously inoculated into a thoroughbred. This experimental animal was kept under strict quarantine and care. No symptomatology of infectious anemia was obtained, however, by this means. After a period of 38 symptomless days, this same thoroughbred was inoculated with five ml. of serum from an acute case. In 8 days, a fever rise was manifested in this experimental horse and continued on with all the classical signs of acute infectious anemia. This was our source of positive serum along with sera from natural infection cases as they occurred in the area.

A film follows which illustrates the three forms of the disease. It was felt that film showing the clinical phases might interest the large animal practitioners. The film demonstrates the following:

1. A natural infection of a 5-year-old mare with a very acute involvement.
2. A chronic case in a 5-year-old mare that acquired the disease through natural sources.
3. The experimentally infected thoroughbred, before and after injection of virus, that developed an acute attack. The typical symptoms shown herein were filmed 62 days after this animal was inoculated with serum from a mare viewed later in the film.

The film attempts to illustrate some of the rather consistent symptoms seen in this disease, as follows:

1. An unusual weakness in the hindquarters with the resultant abnormal gait.
2. Edema of the extremities and the thoracic region.
3. Anemia of the mucous membranes as well as ecchymotic hemorrhages.
4. Characteristic watery blood when viewed grossly.
5. A fairly good appetite even in the last stages of disease when the temperature elevation is extreme.
6. Severe emaciation and general loss of condition of the animal.

The film further shows the necropsy findings such as:

1. Anemia of the tissues.
2. Edema of the subcutaneous tissues with typical icteric coloration.
3. Severely enlarged spleen.
4. Engorged liver in the acute type.
5. The so-called "nutmeg liver" in the chronic type.
6. A severe fibrinous pericarditis is seen in the acutely infected experimental animal.

## HISTOPATHOLOGIC CHANGES OF EQUINE INFECTIOUS ANEMIA

### I

Tissue from the liver, spleen, lung and heart of three horses designated as V3, V4, and V5 who exhibited typical symptoms of equine infectious anemia (naturally acquired) were examined microscopically. The findings were similar in all cases, except that quantitatively they were more pronounced in V4.

In the liver, there was an infiltration by mononuclear cells in the portal and central areas, throughout the sinusoids, and sometimes in the lumens of veins. These cells were lymphocytes, plasma cells and histiocytes (reticulo-endothelial cells). Occasionally, they formed nodules with destruction of liver cells in the area. Hemosiderosis was prominent throughout. Eosinophiles and neutrophiles were also present in smaller numbers. Slight to moderate cloudy swelling of the liver cells was observed.

In the spleen, the lymphoid follicles were atrophic. There was a widespread distribution of lymphocytes, plasma cells, histiocytes and some eosinophiles. Hemosiderosis was slight to moderate. In one animal (V5) there were foci of necrosis.

In the lung, there was congestion and edema. In the alveolar walls and in some areas peribronchially there was an infiltration by lymphocytes, plasma cells, histiocytes and eosinophiles. Hemosiderin granules were found in histiocytes and free in the interstitial tissue.

No significant changes were observed in the sections of heart.

### II

The same tissues were examined microscopically in two horses (V-Dan and V-Duck) in whom the disease was produced experimentally by injection of sera from V3, V4 and V5.

The changes were similar to those already described. Quantitatively, the changes in V-Duck were more pronounced than in V-Dan.

### III

In another animal, "the gray mare", in whom the disease developed naturally and was of an acute nature, the changes were as follows:

In the liver, the central areas of the lobules showed marked necrosis of the liver cells, with cloudy swelling and fat metam-

orphosis of adjacent liver cells. Infiltration by lymphocytes, plasma cells, histiocytes and some neutrophils was present in these areas, in sinusoids, in portal areas and in lumens of veins. Hemosiderosis was prominent.

In the spleen, the capsule was thick, fibrous and wrinkled. The lymphoid tissue was atrophic. Hemosiderosis was slight. There was reticulo-endothelial (histiocytic) proliferation and moderate hemosiderosis of the lymph node.

In the alveolar walls of the lungs there was increase cellularity, with an infiltration by lymphocytes and histiocytes and a significant degree of hemosiderosis. The cells were present in vessel lumens also.

Interstitially in the heart, edema and infiltration by lymphocytes, histiocytes and neutrophils were observed. Sections of striated muscle showed no significant change. Edema was marked in sections of the skin, especially in the lower dermis.

There was prominent cloudy swelling and autolysis in the kidney sections. There was thickening of basement membranes in glomerular tufts and possible increase of its cells. Hemosiderin granules were present in small quantities in some glomerular tufts and interstitially. Foci of infiltration by lymphocytes, plasma cells and histiocytes were noted in areas.

In multiple sections of brain, no lesion was observed.

(Note on "the gray mare": The changes, especially in the liver, are compatible with those of acute, equine infectious anemia. The possibility that some of the central necrosis of the liver was the result of chronic passive congestion is to be considered. There was non-specific, interstitial myocarditis which could have resulted in cardiac failure with passive congestion).

#### IV

Tissues from the horse "Pliant", in whom the disease was induced experimentally, were examined microscopically:

In the liver there was a pronounced infiltration by lymphocytes, plasma cells and histiocytes in central and portal areas, throughout the sinusoids and in venous lumens. Hemosiderosis was abundant.

Hemosiderosis was also abundant in the spleen and lungs. Foci of hemosiderosis were noted in the glomeruli and interstitial tissue of the kidneys, and in the lymph nodes.

Other changes were: Cloudy swelling of renal tubules; thickening of basement membranes and increased cellularity of the glomerular tufts; foci of infiltration by lymphocytes

and histiocytes in the kidney and lung interstitially and in lumens of some vessels; lymphoid hyperplasia of lymph node; and edema, with focal infiltration by lymphocytes and histiocytes, in the myocardium and subendocardium.

## V

Tissues of five other animals ("Filly", "Palomino", "Pal", "Pinto" and "Close"), said to have symptoms of the natural disease, were studied. It should be noted that the disease was not proven in these cases by transmission to other animals. In four of them the changes generally were similar to those described above in the horses of the V-group. In addition, the bile ducts and canaliculi of the liver of one of these ("Palomino") contained plugs of bile pigment.

The 4th horse ("Close") was of particular interest. He had had symptoms only a very short time. The inflammatory cell infiltration and hemosiderosis of the liver and other organs were slight. In the liver there were moderate cloudy swelling and fat metamorphosis, and bile pigment in the bile ducts and canaliculi. An interesting feature, not seen in the others, was the presence of round, acidophilic masses in the cytoplasm of some of the liver cells; sometimes the masses were extruded into the sinusoids. These cytoplasmic, hyalin bodies are reminiscent of the acidophilic bodies in livers of viral hepatitis and the Councilman bodies of yellow-fever in human cases. Further study is being made to determine their significance.

In this latter horse, the kidneys showed rather pronounced degenerative change (cloudy swelling, hydropic degeneration) and even individual cell necrosis. The glomerular tufts in this horse and in two other horses of this group ("Filly" and "Pinto"—The only ones in which kidney sections were available) showed thickening of the basement membranes and increased cellularity. These changes, similar to those described above in "the gray mare" and "Pliant", may be significant and resemble those of membranous glomerulonephritis. More cases with renal lesions, together with more controls, must be studied to determine the significance of these changes. Also in the kidneys of "Close" were foci of cells and hemosiderosis as previously described. Hemosiderin pigment was present also in the lung.

Study of the bone marrow of all of our animals is not complete at this time. In one animal ("Filly") with the natural disease, there was hyperplasia of the marrow.

The brain was examined in only one of these horses ("Close"). It showed no lesions. Heart sections were available in three horses. In two there was focal, non-specific myocarditis ("Pinto" and "Filly").

## EXPERIMENTAL APPROACHES TOWARD A LABORATORY DIAGNOSIS

The approach to the development of a laboratory method for aid in the diagnosis of Equine Infectious Anemia has been, thus far, fairly well restricted to the use of chick embryo tissues. To some degree, three types of procedures have been studied, as follows, with these tissues:

### A. Chick Embryo:

Following much after the pattern of studies conducted by Dreguss and Lombard at the University of Pennsylvania and the Grayson Foundation, 6-10 day embryos were inoculated by three routes; yolk sac, allantoic and amniotic cavities. The serums were used from five horses with clinically positive evidence of viral anemia. As control, serum from a young horse with no history of past illness was used. In each case an inoculum of 0.5 ml. serum was injected. Approximately 300 egg embryos were studied for each route of injection and an additional 300-400 eggs for the normal control serum. Serial passages of infected egg material were also attempted in an effort to establish evidence of virus multiplication or rise in hemagglutination titre. Our results were practically identical to the findings described by Drs. Dreguss and Lombard. The death rate in the embryos inoculated by clinically positive sera was only 3-4% higher than the embryos injected with normal horse serum. Most of the embryos that did not survive in the positive serum series showed a marked swelling and redness of the skin with diffuse hemorrhages. On one occasion, however, the same gross pathology was found in one embryo that died from the normal horse serum inoculation series. The presence of hemagglutination was also verified but no increase in titre could be demonstrated by serial passage. After two or three passages, no hemagglutination could be demonstrated. It was also impossible to find any increase in virulence of virus by these techniques. After using over 1,200 embryo eggs, it was felt that this approach yielded results which were inconsistent and often impossible to reproduce.

### B. Chick Embryo Transplants:

It was felt that a chick embryo in its very early development might be more susceptible to the anemia virus action. Chick embryos in a 2-6 somite stage were aseptically removed from the egg by cutting the germinal disc away from the egg and transplanting

this disc onto a nutritive agar medium. The nutritive medium is composed of egg albumin, balanced salts, etc. A test serum can be incorporated into the medium and the influence of such serum on the growth and development of the embryo is easily discernible. Under controlled conditions a transplanted embryo can live and develop normally for a period of 4-6 days.

Serums from clinically positive horses were tested by this procedure and their influence on the developing embryo was evidenced by alterations in the head and thoracic regions within 48 hours. The negative control serums did not alter or modify the normal development of the embryo.

To enhance and simplify the test procedure, 24-hour embryos were inoculated "in ovo" by cutting a window in the shell and injecting 0.05 ml. of test serum directly under the embryo or germinal disc. The shell windows were resealed and incubated for 24 hours. The germinal discs were then removed from the eggs, fixed in Bouin's Solution, mounted and stained. Both serial cross-sections and whole mounts were studied. Serums from naturally acquired viral anemia, as well as experimentally infected horses caused widespread abnormalities in development, with gross involvement of the neural tube, limb bud, and heart. Histologically, marked involvement of all three germ layers was observed, with mesodermal derivation being primarily affected.

#### C. Chick Embryo Heart Tissue Cultures:

One millimeter fragments of 8-10 day old chick embryo hearts were grown in culture by the double coverslip method. The transplanted tissue mass soon flattens and a sheet of fibroblast cells grow out in all directions within 48 hours. Following a wash process, these cells were then subjected to the influence of horse test serum. A second method involved the growth of fibroblasts directly in horse test serum.

The horse test sera studied were from normal horses, naturally infected horses, and experimentally induced infection horses.

The chick heart fibroblast cells, when incubated for 48 hours with positive sera, demonstrated an excessive accumulation of fat vacuoles. Apparently, the lipid metabolic processes of the chick heart fibroblast cells were disrupted, or interfered with, when exposed to sera from clinically positive viral anemia horses. Fibro-

blasts grown in normal horse serum develop small scattered fat globules, after 48 hours, which are readily washed out in the feeding process. The positive horse sera included large numerous fat globule formation which did not wash out. This investigation is continuing with many other tissue culture cells; human amnion, HeLa, horse tissue cells, etc. It can be stated at present that cytomorphologic changes have been seen in amnion cells when exposed to positive horse sera. It seems apparent to us that chick and other tissue cells can be used to demonstrate a reaction to anemia virus serum. What the significance of these cellular alterations may be is problematical. The primary concern at this time is to establish some concept of the specificity and sensitivity of these cellular reactions. Since horse inoculation is the only certain method for obtaining evidence as to the presence of the infectious anemia virus, sera yielding cytomorphologic changes must be verified by horse studies. Only then could any reasonable interpretation of these tissue culture cell reactions be made.

The hematologic studies done in the natural and experimentally infected animals are identical with those reported by the Grayson group. Also electrophoretic patterns show the changes described in their monograph. It is felt these changes are good aids in confirming the clinical diagnosis but not specific. Possibly a specific test will materialize as the studies continue.

# SURGICAL TREATMENT OF TENDINITIS

*By*

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I heard a story last evening that tickled me. It seems that this girl was very fond of liver and eggs. So she went to the grocery store and bought a pound of raw liver and a couple of fresh eggs. When she walked out of the store, the bag slipped and the whole mess fell on the sidewalk. She was standing there looking at this rather disagreeable conglomeration when this drunk came up to her and said, "Don't worry, Lady, it wouldn't have lived anyway. Its eyes were too far apart."

The reason I tell that story is because since arriving in Chicago I have been standing here wondering what has happened to my slides! I had about 20 Kodachrome slides. I wasn't quite satisfied with them so I sent them back for some processing. That was 16 days ago and as of yesterday they weren't at the place where I sent them and they weren't in Lexington. So I think that they are the victims of the Christmas rush in the mail.

This business of surgical approach to bowed tendons—I'll go back and give you a little history of the procedure and my interest and approach to it. The first time that it ever occurred to me that one of our profession could go in and open up the complete flexor tendons of a thoroughbred animal and hope to have him go back to racing, was at a meeting of the Kentucky Racing Association last December. Dr. Art Davidson was kind enough to report that he had advised two orthopedic surgeons, Drs. McKeever and Pitkin, on an operation for the relief of tendinitis some time during the previous fall. He thought it was a pretty heroic approach, and the rest of us knew it was. The report stuck in my mind, and in a discussion with Dr. Lyman Smith, an orthopedic surgeon, in March, 1957, I brought out the tremendous need for such a surgical approach. I felt that only about 10 to 15% of the bowed horses actually returned to the races in anything like their former economic value. Under the ordinary treatment, which is usually firing, and blistering either internal or external, the ordinary horse is laid up anywhere from six months to a year and a half.

A brief visit to any breeding farm will disclose that more horses are probably retired from racing from bowed tendons

than any other single type of pathology. And that applies almost equally to the standardbred as the thoroughbred. You all remember that disastrous spring when Dark Star and Turn To, a couple of hundred-thousand dollar horses bowed their tendons and were retired to stud right at the most promising time of their racing career.

Actually I felt that we had no effective treatment to offer. In fact every time you spoke to an orthopedic surgeon about a bowed tendon, you immediately were on the defensive because, as I say, the only thing you had to offer was rest and counter-irritation, some type of cauterization or chemical treatment. None of them were particularly effective. Another thing that I am ashamed to admit but that is true, is that as far as I know neither I nor any other veterinarian had a very clear conception of the pathology involved in a genuine bowed tendon. That seems strange, but nevertheless it is true. Horses don't die of bowed tendons and if anybody had ever taken time out to dissect a bowed tendon and if it is in the literature, it was never impressed on me.

Dr. Smith described to me a medical technique referred to as the "Snap Thumb Operation." It has been done on thousands of humans successfully and he felt that the condition and pathology involved in man was analagous to bowed tendons in equines. The original technique of surgery was worked out by Dr. Smith after he had heard of the McKeever and Pitkin efforts. Since then no less than five orthopedic surgeons have observed and suggested aids and demonstrated techniques which have gradually amalgamated to the basic operation I now shall describe. In addition to the orthopedic surgeons some twenty-one veterinary surgeons have observed and suggested minor modifications in technique. These men have literally come from Canada to Puerto Rico and from New York to California. In other words there was a pronounced and deep need in veterinary medicine for some surgical approach to tendinitis. The operative technique is truly a result of a meeting of qualified minds of the medical and veterinary service. To me it is an example of the progress that can come through cooperation of the veterinary and medical professions.

Three fundamental facts stand out from the experiences that we have gained from doing surgery on these tendons:

1. Complete asepsis makes the veterinary surgical technique heretofore considered impossible, a routine procedure.
2. There is no reason why a 12" wound won't heal just as fast as an inch and a half wound. I beg of you don't hinder your efforts by working through an impos-

sibly small opening because the sum total of your efforts will simply be to traumatize the edges of those wounds and you won't get good healing.

3. The fact which excited me intensely and gave me reason to hope that this method would be successful is the fact that the pathology of the bowed tendon in no way affects the tendon structure itself. It is characterized by paratendinous fibrosis.

Since March, 1957, we have performed this operation on a total of 63 animals. The first 5 or 6 animals were done under local anesthesia and in a standing position. It became apparent to me that I couldn't do as good a job under local anesthesia and acrobatic surgery, as I could under sedation and control and we shifted the site of our operations to the table and now we routinely do this operation under local anesthesia plus sedation.

The average bowed tendon can be classified as to whether it is high or low. If you will notice the diagram on the board, I think a tendon that is more or less bowed as I have it demonstrated there by the outside line right up under the inferior check ligament and down to the top of the upper end of the synovial structure, you call the high bow. The average low bow involves the area from about the middle of the cannon bone down to and including the structures of the fetlock itself. So we arbitrarily, due to location, classify tendinitis as high bow or low bow. Invariably they are also classified as acute or chronic. In my particular practice which is not on a race track I don't see as many acute tendons as some other veterinarians. The majority of the tendons presented to me for surgery have been chronic. Some of them have been fired, turned out for 6 to 8 months or a year, broken down, blistered, turned out again, and broken down the third time. Another classification, in my own notes that I have kept on these cases, refers to them as slight, moderate or extensive. The terms slight, moderate and extensive merely indicate how much dissection is necessary to free the structures of the flexor structures. The more of these tendons I worked on the more I became aware that some fairly meticulous dissection was in order. In order to do that dissection properly and with a minimum of trauma I felt that it was necessary to put these animals on the table, at which time I could break the structures properly and do the operation under complete asepsis.

If you will examine diagram taken from Sisson; the structure here that we are particularly interested in is the deep flexor tendon. I will indicate the spot where the superficial flexor tendon makes its encircling band around the deep flexor. This is the volar annular ligament. This is a schematic diagram and in practice I think you will find that the upper border of the flexor

annular ligament is well above this area where the superficial flexor begins to form its ring, and the digital annular ligament as such is not a distinct structure. In other words you make your incision down to the volar annular ligament and digital annular ligament. The technique of the operation will vary according to that part of the flexor tendons that are involved. I actually had one horse that had a high and low bow with constriction between. He was presented to me with the compliments of Dr. Riley of Michigan State College. My farm manager took one look at the horse and said, "Say, I thought that guy was a friend of yours."

I think that the prognosis depends on the amount of pathology that is present. The slight low bows can best be relieved by the incision plus the cutting of the annular ligaments, plus the opening up of the superficial digital ring. In some of these brand new conditions there is no prognosis as such and you can just go in there with a sterile 4 by 4 swab and wipe out the extruded synovia that is incorporated under the annular ligament and ring of the superficial digital flexor. In some old chronic hambone bows, the pathology actually starts right under the accessory carpal bone. The other day I did one in which I had to go in and actually remove the excess synovia that lays around and in behind the inferior check ligament. We made our incision through the lateral posterior aspect of the leg, reflected the skin, carefully reflected the most superficial layer of fascia and then proceeded to remove all the adhesions, and all the fibrosis that lay in these layers of fascia which cover the superficial and deep digital flexors and seem to fit in just behind your volar arteries and nerves. The height of your incision should be limited. I find that I get in trouble the minute I go up back of the knee. When the knee flexes, it tends to push down your bandage and the keynote to the success of this surgical procedure is pressure bandages following surgery. The minute your pressure bandages drop below the top of your incision, no matter if you are completely aseptic and even though you have healing of 7 to 8 days extent, edema will occur immediately. Your incision will frequently break open. I am referring specifically to these conditions of extremely high bow where you have to go in well up under the knee to reflect that excess fibrosis.

Gentlemen, in a nutshell, that is it. Carefully expose your structures by minute dissection and free the superficial digital flexor and digital deep flexor from all the fibrosis; peritendinous fibrosis. Some times you remove in really bad bows a piece of fibrous tissue that is 12" long,  $\frac{1}{4}$ " thick, and 3" wide. The leg looks considerably better when you've gotten rid of that fibrosis. Strangely enough the animal is not acutely lame. They are skin

sore, but they will walk practically sound within 4 or 5 days. The more extensive your dissection and the more blood vessels that are not properly tied off, the more your post operative edema and the less chance you have of first intention healing. But I do feel that it is paramount that you have complete asepsis. I do like to get complete healing with no drainage, and it happens in by far the greater majority of cases, about 85%.

Here again we have another schematic diagram. It shows all of the superficial portion of the leg reflected away. This is the cut edge of the annular ligament. The digital annular ligament is still in place. As I say, in my surgery I don't find any definite place where the annular ligament stops and the digital starts. It all seems to be continuous, but the incision is carried down on the lateral posterior aspect of the fetlock.

Your first structure to open is the tendon sheath. If it is an acute tendinitis there is a tremendous welling out of synovia. I feel that blood-fused synovia indicates probably a poor prognosis. Then I very carefully cut the ring of the superficial digital flexor. In a brand new bow where there is not extensive fibrosis, that is all the surgery involved. Close your skin, and leave these cut edges free. Some people have taken a segment out of a ring of the superficial flexor. In an extreme low bow, it is almost imperative that you remove the entire annular ligament of the fetlock. You can't separate the fibrosis from the annular ligament, you just have to take the whole thing out. And it is in those cases where I use my superficial layer of fascia to complete a closure so that you don't get a lot of synovial leakage. I use a two layer closure in those cases.

As I mentioned a while ago, the post operative care consists of tetanus-antitoxin, and antibiotics (I carry those on for a 10-day period). I use a pressure bandage, and recommend regulated exercise. Now I think that is imperative. We have gone in there with the express purpose of removing fibrosis and adhesions and unless you keep that animal exercised, *and regularly*, and at more than a walk, I feel the whole operation is doomed to failure. I think that post-operative regulated exercise is probably the most important single thing in the future success of the whole procedure. We hand-walk them for 3 to 4 days, put a saddle on their back or if they are a standardbred we hook them, and we let them go right on to their taste. The minute a horse walks flat footed, we ask him to jog a few steps. When he jogs sound, we ask him to hit a little canter. Some of them will gallop out as early as a week after the operation. Inside of 60 days, the average animal is galloping 2 or 3 three-minute miles, or if he is a trotter, it is probably a four-minute lick. We try to not take them out on an uneven surface. Nor do we give them a chance to slip or slide or put extreme

flexion on the structures involved. But regulated exercise for 60 days post-operatively I think is an absolute necessity.

Lots of people have gone in on these acute bows, wiped them out so to speak, opened up the structure, put them back together and these animals will be back galloping within 2 to 4 weeks, and racing within 90 days. Personally, having had a few operations myself, and many sutures, I know that these old scars are sore for months thereafter. I think these animals are pretty prone to have skin soreness. The thing that terrifies me, and I have had it happen a couple of times, is that those animals stride out fairly naturally until they hurt themselves and/or there is a sudden pain. Then they put undue stress on the other leg. Now out of this series of 63 animals I have had 2 come back and bowed the good leg. Following the 60 days of regulated exercise, it usually takes the average thoroughbred man 90 days to get a horse ready to run  $\frac{3}{4}$  of a mile. So I think that in the majority of cases you can look your client in the eye and tell him that if all goes well, his animal will be ready to run  $\frac{3}{4}$  of a mile within 5 months after the operation. I think that is all the time they really need. The reason I think it is imperative to give them that much time is because it takes that long for the cellulitis that results from the healing process to go back to normal and for the leg to assume a normal appearance again. I am speaking not of the low acute slight bow. I am speaking of the average one you run into that has probably been tried, broken down, blistered and turned out and then he goes sore when they take him up the second time. As I mentioned earlier I have done 63 of these. The majority were thoroughbreds; there was one jumper in the outfit, and there were 12 standardbreds. Four of these standardbreds had bows on both front legs. One thoroughbred had bows on both front legs. Both of them were subjected to surgery.

The one horse that we operated on both front legs placed in an allowance race the other day in Maryland. I have had one winner, and one other horse to place. It is awfully hard to keep track of these horses: I am not absolutely positive about this. I enlisted the aid of Mickey McGuire, columnist for the Racing Form, to give me a hand. He agreed to take the horses names and the owners and not divulge them as such, but to help me to come to some intelligent conclusion on the statistics of these type of operations. I hope that with the help of some of you gentlemen who have been doing this operation to be able to put in the literature and classify these tendons intelligently, and be able to give some concrete prognosis on the operation based on statistics of a substantial number of cases.

Summing up my feelings on the thing, I feel that an operative technique is the only way to remove the adhesions, the

peritendinous fibrosis that accompanies the so-called bowed tendons.

I feel that the animals tolerate surgery better than firing or blistering.

I feel that if everything goes well you can save the average owner approximately 6-months board bill. In other words, I think you can accomplish in 6 months what firing or blistering, assuming that is successful, will accomplish in a year.

I feel that only 10 to 15% of the animals that bow and are treated under the ordinary methods go back to racing according to their previous form.

Lastly, I think that this is a highly professional and logical approach to the problem, one which we don't have to apologize for in any way. I don't want to go out on a limb and say that this is the best treatment at present for bowed tendons. I don't think I have to. In another 6 months we will have done enough of these operations that this procedure will either stand or fall on the results that are apparent to everyone.



# ETHER ANESTHESIA IN HORSES

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Effective and controlled anesthesia in large animal practice has been the deterrant to many indicated surgical procedures. Despite the development of many new anesthetic agents, the veterinarian concerned with surgery in large animals has been handicapped because of inability to, 1) properly control the degree of anesthesia, 2) rapidly change the degree to either a higher or lower level as indicated, and 3) effect almost immediate post-surgical recovery.

The latter is of great importance, 1) to the welfare of the patient, 2) to avoid prolonged period of restraint, and 3) to reduce the number of people required to bring the animal out of anesthesia. Perhaps most important consideration of all is the safety of recovery to the patient. Depth of anesthesia in the barbituate group of drugs such as Surital Sodium and Pentathol Sodium and even Nembutal, has been very satisfactory; however, the recovery from these agents used singularly or in combination with other anesthetic agents has resulted in varying degrees of swimming and struggling, even to self-destruction on the part of the animal. This, of course, has limited the usage of these drugs for anesthetic agents. The disadvantages of unfavorable anesthetic agents has prompted the consideration of gas anesthesia which acts more favorably for the patient as well as for the operator.

A brief review of inhalation anesthesia may be in order. A volatile anesthetic affects the nervous tissue by a reversible physical union with vital cellular substance. It is in a state of gaseous equilibrium. A volatile anesthetic is effective by several routes of administration, provided it reaches the blood, but it is best given by inhalation. In the alveoli of the lungs the anesthetic gas diffuses through separating membranes into the circulating blood of the lung capillaries. The gaseous anesthetic as it dissolves in the blood stream is transported over the body to lipid tissue into which it diffuses as a result of greater solubility in fats than in any other medium. The central nervous system contains more lipoids than any other part of the body and, therefore attracts far more anesthetic than any other system. In addition, the central nervous system is highly vascular so that proportionally more anesthetic is carried to that part of the body.

At rest average figures for the volume of blood per 100 grams of tissue are; for the central nervous system, 160 milliliters per minute, the intestines 60 milliliters per minute; and the legs, 10 milliliters per minute. Obviously the central nervous system is more susceptible than other systems to the effects of the anesthetic because of its high vascularity and lipid content. On the other hand, when administration ceases the anesthetic will diffuse out of the brain more quickly than other tissues with a smaller blood supply. The depressant effect of ether is due more to its concentration than to the quantity. It has been frequently demonstrated that twice as much ether is required to produce anesthesia at a low concentration as is required at a high concentration of inhaled vapor. Therefore a moderately high and effective concentration of ether is desirable for the production of surgical anesthesia. The excretion of a volatile anesthetic involves the same diffusion processes as were active during administration except that they operate in the reverse direction. The rate of excretion of a volatile anesthetic is dependent upon proper venilation of the lungs. Anesthetic concentrations of ether do not effect the heart. In this respect has a great advantage over chloroform because there is no danger of inducing ventricular fibrillation. Generally speaking ether is the safest of all general anesthetics. Ether satisfactorily depresses the central nervous system without undesirable effects upon other systems of the body. It permits ready control of the level of the anesthesia. Another advantage of ether is that it is inexpensive. Ether also has the advantage of being a particularly effective muscle relaxant.

To permit usage of ether in a horse several different units were designed, modified, and some discarded as various technical problems were presented. The apparatus that we are now using is a simple one. It is simple to operate and has been quite effective. Utilization of a semi-closed system with calibrated blow-off exhaust and rebreathing through a carbon dioxide absorber permits the use of a minimum quantity of ether with maximum control. The equipment consists of a series of four vaporizers which are all connected permanently to a single ether chamber. Each of the vaporizers is controlled by means of needle valves which are mounted on a single manifold and are activated with a pressure of 40 P.S.I. Oxygen passes from the cylinder and the regulator, or, from a source of piped oxygen by means of a pressure hose through a master needle valve which is provided as an added safeguard so that the entire system may be closed off at any time. The outlet duct of each of the four vaporizers is connected to a manifold by means of a corrugated flexible hose to allow for ease of mobility. The four inlets in turn connect to a common duct consisting of a 3" flexible hose leading to the inspiratory T. The distal end of the

T comprises a Soda Lime canister for absorption of carbon dioxide. The terminal section of the canister receives a 60 liter bag with its tail inlet for administration of pure oxygen through 4' corrugated oxygen hose taken from the oxygen manifold supplying the ether vaporizer. The proximal end of the inspiratory T is again fitted with a 3" flexible rubber tube which can be fitted either with a anatomical equine mask or endotracheal inflatable cuff catheter. The mask is a lightweight aluminum welded unit with a removable inflatable rubber cushion. This inflatable rubber cushion forms a seal between the horse's head and the mask which prevents the escape of ether during anesthesia. When the endotracheal catheter is used a catheter adaptor is provided to fit into the 3" flexible rubber tube. The system which we have followed to use this equipment has been adapted to our individual needs and other equipment. We have used various pre-anesthetic medications primarily to diminish apprehension. When we are about to anesthetize the horse we have used a suitable dosage of succinylcholine chloride which enables us to completely control the horse on the operating table, which in our case, is a hydraulic table coming out of floor level. The mask is then put on the horse's head and the oxygen is turned on. And all four vaporizers are turned on by turning needle valves so as to fill the flexible tubes and the breathing bag with a dense vaporized ether oxygen mixture. Usually complete surgical anesthesia may be expected in from 10 to 15 minutes with no resistance on the part of the animal. By surgical anesthesia we expect a completely reflexless anesthesia. From this point on the degree of anesthesia at which the animal is maintained can be readily and instantly controlled by either increasing or decreasing the flow of oxygen through the needle valves. If at any time during induction of anesthesia or during surgery immediate high volume of ether mixture is desired, the adjustable top of the ether vaporizers may be raised. This causes a large influx of room air to be drawn into the vaporizers and greatly increases the velocity of concentration of ether vapor. These same valve tops are fitted into vaporizers so that in the event that if excess pressure should build up in the unit, these valves will open and allow the surplus to blow off. As the surgery approaches completion it is possible to cut down on the flow of ether vapor to lighten the plane of anesthesia. As the flow of ether oxygen is shut down, it is further advisable to turn on the oxygen needle valve which will result in the administration of pure oxygen to the animal. The exhausted air which tends to build up the pressure will blow off at the vaporizer valves. As the animal begins to regain consciousness it may be further advisable to hold the mask lightly to the face so that the exhaled air would escape easily.

The administration of oxygen before and after surgery is

At rest average figures for the volume of blood per 100 grams of tissue are; for the central nervous system, 160 milliliters per minute, the intestines 60 milliliters per minute; and the legs, 10 milliliters per minute. Obviously the central nervous system is more susceptible than other systems to the effects of the anesthetic because of its high vascularity and lipid content. On the other hand, when administration ceases the anesthetic will diffuse out of the brain more quickly than other tissues with a smaller blood supply. The depressant effect of ether is due more to its concentration than to the quantity. It has been frequently demonstrated that twice as much ether is required to produce anesthesia at a low concentration as is required at a high concentration of inhaled vapor. Therefore a moderately high and effective concentration of ether is desirable for the production of surgical anesthesia. The excretion of a volatile anesthetic involves the same diffusion processes as were active during administration except that they operate in the reverse direction. The rate of excretion of a volatile anesthetic is dependent upon proper ventilation of the lungs. Anesthetic concentrations of ether do not effect the heart. In this respect has a great advantage over chloroform because there is no danger of inducing ventricular fibrillation. Generally speaking ether is the safest of all general anesthetics. Ether satisfactorily depresses the central nervous system without undesirable effects upon other systems of the body. It permits ready control of the level of the anesthesia. Another advantage of ether is that it is inexpensive. Ether also has the advantage of being a particularly effective muscle relaxant.

To permit usage of ether in a horse several different units were designed, modified, and some discarded as various technical problems were presented. The apparatus that we are now using is a simple one. It is simple to operate and has been quite effective. Utilization of a semi-closed system with calibrated blow-off exhaust and rebreathing through a carbon dioxide absorber permits the use of a minimum quantity of ether with maximum control. The equipment consists of a series of four vaporizers which are all connected permanently to a single ether chamber. Each of the vaporizers is controlled by means of needle valves which are mounted on a single manifold and are activated with a pressure of 40 P.S.I. Oxygen passes from the cylinder and the regulator, or, from a source of piped oxygen by means of a pressure hose through a master needle valve which is provided as an added safeguard so that the entire system may be closed off at any time. The outlet duct of each of the four vaporizers is connected to a manifold by means of a corrugated flexible hose to allow for ease of mobility. The four inlets in turn connect to a common duct consisting of a 3" flexible hose leading to the inspiratory T. The distal end of the

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completed eliminates the necessity of keeping the animal on the floor for long periods as is necessary when barbituates are used intravenously. Administration of oxygen allows a speedier recovery and permits the animal to stand on its feet more readily than without administration of oxygen. This also eliminates most of the violent swimming and thrashing accompanying the excitement stage as found in barbiturate anesthesia. The use of ether anesthesia has allowed various types of surgery to be performed that we feel would be extremely difficult under ordinary circumstances. It might be well to mention here that ether anesthesia in horses is currently being used in Germany, Scotland, and England with a considerable degree of success.

It would be natural to anticipate the question of safety to the operator with the usage of ether. Dr. Dripps, who is the head of the anesthesia department at University of Pennsylvania, has assured us that the described method of ether anesthesia is safe from explosion. One other question which naturally arises is the possibility of gastric disturbances in horses with ether anesthesia. To date, we have had no reactions of any sort.

In conclusion we offer the described procedure of ether anesthesia in horses, either alone or in combination with other anesthetic agents.

## SPEECH BEFORE OPEN FORUM

MR. MARSHALL CASSIDY

Executive Secretary of the Jockey Club

Mr. President, Officers and Members of the American Association of Equine Practitioners and Guests:—

I feel something like a fish out of water here among you learned men of the veterinary profession. However, I am very honored to have been invited to speak to you and I hope I may be able to assist you in any problems that may have developed in the past several years, particularly in respect to racing regulations as they may relate to drugs of a possible stimulating nature.

Because of reputed statements by members of various associated groups, feelings have been hurt and resentment has grown. I am sure all of us here today will make every effort to achieve the harmony and understanding which will advance our mutual interests and benefit racing as a whole.

First I would like to review briefly the incidents of the past half century that I think may have some bearing on the present unsettled condition. I am, of course, speaking of services aside from the actual treatment of diseases and injuries of the animal.

In the early quarter of this century there was a rather common and well-known practice among horsemen to seek some aid for their horses to assure a successful campaign. At that time there were no specific rules or regulations prohibiting the administration of so-called minor stimulants. Brandy and coffee may have been the most common ingredients given, but heroin was suspected in rare cases. As there were no scientific tests to determine the presence of a stimulant and to prove that anything of such a nature had been administered, the official veterinarian was called upon to render his professional opinion from observation of physical symptoms such as dilated pupils, fast heart action, nervousness and difficulty for the horse to cool out after his race.

I remember there were even a couple of instances where the official veterinarian was sued by the owner of an animal that had been adjudged stimulated and had therefore been penalized by the Stewards.

During these years any trainer who was unusually successful was suspected of having a good prescription that made his horse run faster and further without showing the recognizable signs of stimulation.

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Therefore, for that period anyway, a veterinarian was in the unhappy position of either being censured for judging an animal stimulated or being suspected of having provided a trainer with an unfair advantage over his competitors by means of a special concoction.

About 1932, Dr. Enslinger, Commissoiner of Narcotics for the Federal Government, in making a drive throughout the country to suppress the sale and usage of narcotics, made several raids on race tracks and found in a relatively few instances the presence of such drugs in the medicine cabinets in the stable area.

Unfortunately, this resulted in a nation-wide scandal and frantic efforts were made to clean house and start afresh under better supervision. Mr. Joseph E. Widener, then vice chairman of The Jockey Club and president of Hialeah, sent Dr. James G. Catlett, who is now chief veterinarian for The Jockey Club, and Charles Morgan, who is now chief chemist for the New York State Racing Commission, to France to study the new saliva tests in use there to determine the presence of medication. After they returned to the United States and proved the value of the tests here, various state racing commissions promulgated rules making it mandatory to suspend the trainer of any horse found to have been administered a stimulant, whether or not the trainer himself had any knowledge of the act.

At that time the laboratories were able to find evidence only of narcotics and very few other drugs, so that the unscrupulous trainers necessarily sought prescriptions that contained those stimulants which could not be detected. I would not doubt that nearly every veterinarian working in racing was approached hundreds of times for just such a prescription. Every time a horse showed sudden improvement and won a race and was not subsequently involved in a stimulation ruling, the trainer was suspected of having found just the right ingredient.

As the years went by our chemists and our veterinarians found ways and means to detect more and more individual drugs but racing authorities were then confronted with a problem that so far no one has solved. What constitutes a drug that may affect the racing condition of a horse? Are tonics to build up health to be so classified? Are hormones and vitamins to be included? If so, what about the secretions of the glands of the animal which can be administered as a stimulant?

And last but not least, what about icing a horse's leg? Does that not affect his racing condition?

After years of trying we finally have secured a change in the rules in some states that permit fair judgments to be made. I think our rule in New York is a good one. For the benefit of

the veterinarians here today who may not be familiar with the rule I would like to read it.

Quote. If the Stewards shall find that any drug, stimulant or narcotic has been administered or attempted to be administered, internally or externally to a horse before a race, which is of such a character as could affect the racing condition of the horse in such race, such Stewards shall impose such punishment and take such other action as they may deem proper under any of the Rules, including reference to the Commission, against every person found by them to have administered, or to have attempted to administer or to have caused to be administered, or to have caused an attempt to administer, or to have conspired with another person to administer such drug, stimulant or narcotic. The trainer, groom and any other person having charge, custody or care of the horse, are obligated properly to protect the horse and guard it against such administration or attempted administration and, if the Stewards shall find that any such person has failed to show proper protection and guarding of the horse, they shall impose such punishment and take such other action as they may deem proper under any of the Rules, including reference to the Commission. Unquote.

Such a rule helps establish justice in one sense but it does not clarify the problems of veterinarians' service. A veterinarian can prescribe for a sick or injured animal and certify to the racing authorities that the trainer can administer certain medication for a specific time. But in doing that he discloses his own valuable knowledge of medicine. He makes known to his competitors the ingredients in his prescription which, if its results are successful, give him recognition in his field.

The New York State Racing Commission about a year ago, after discussing this problem with the veterinarians practicing in New York, adopted the following rule 94A:

Quote. Every such veterinarian who shall prescribe or use any medication or treatment which contains a drug or drugs which he has reason to believe are of such character as would affect the racing condition of a horse in a race, shall at the time of such prescribing or use deliver to the Steward of the State Racing Commission and the trainer of the horse under treatment a written statement setting forth the names of the horse and of the trainer and the fact that such medication or treatment, as the case may be, contains a drug, stimulant or narcotic which in the opinion of the veterinarian is of such a character as could affect the racing condition of the horse in a race. Unquote.

For a time the veterinarians filed a copy of the statement required. Then gradually these reports diminished and I am sure that during the last six months at least no reports have been received.

In discussing this matter with the veterinarians we found that they had been able to eliminate entirely any medication in their prescriptions which, in their opinion, was of such a character as could affect the racing condition of a horse, as they did not wish to risk becoming involved in an investigation should the presence of such a drug be found in the saliva or urine tests. However, if under this procedure, after a race, a stimulant is found in the body secretions of a horse which had previously been treated by a veterinarian, I wonder whether the veterinarian might be placed in an embarrassing position, especially if the owner, trainer and stable help swore that no medication other than that prescribed had been given to the animal.

I am sure we do not have the perfect solution to this problem so that the rule may operate with fairness to all concerned. Would it be possible to have an accurate statement of the prescription filed, in a sealed envelope, with the Commission? This envelope would not be opened unless the animal for whom the medication was prescribed was found to have been stimulated. Later the sealed envelope could be returned to the veterinarian at the expiration of a set period of time.

For a number of years there has been pressure to include in the rules and regulations a prohibition against the administration, within 48 hours of the time a horse races, of any medication which may affect his racing condition. I have always opposed that and still do on the grounds that it is impossible to determine how long the presence of a certain medication will remain detectable in the secretions of the animal. The statement by the trainer or the veterinarian that the administration of such medicine had been made 50 hours before the race could not be proved or disproved unless our chemists had made a complete test of, and recorded the chemical reactions to all stimulating drugs. In addition to that there is the ever-present danger of an outsider administering a supplementary charge of the same ingredients at a time when it would be very effective as a stimulant to the horse. In this case if the veterinarian had prescribed and administered the drug he might have a difficult time proving that he was not the one responsible for the horse's stimulated condition, as in the investigation following the stimulation there may be no evidence of anyone other than the veterinarian having had access to the animal prior to his race. Because as far as I know we do not have a quantitative analysis of any drug so found in the urine or saliva, we would be unable to determine whether the amount contained in the prescription of the veterinarian was the total amount of a drug given to an animal and we could not absolve the veterinarian of a certain responsibility.

Now that I have reported on the background of stimulation in respect to both administration and detection, I would like to

be specific and tell you that the veterinarians themselves have been, in a way, responsible for a lot of the agitation that is going on throughout the country.

I have had reported to me by reputable stables that veterinarians have tried to charge a fee for treatment of a horse commensurate with the horse's success in a race following such treatment. A veterinarian is reported to have said that he had done as much or more than the trainer who has maybe worked for months to bring his horse up to a race. By making such a statement the veterinarian is spreading suspicion regarding the ethics of his practice. I believe that such procedure should be completely abolished and standard fees charged for services, regardless of racing results.

As long as stimulation is possible, we are going to have suspicion placed upon the veterinarian and anyone else who has a knowledge of medicine and racing. Therefore I think it would be well for the American Association of Equine Practitioners to set hard and fast rules prohibiting anything that may reflect on the profession, and to have the violation of such rules carry a drastic penalty. You have an organization that is of tremendous importance to the racing industry working hand in hand with the authorities. The best interests of horsemen, management and yourselves would be served if your membership included every veterinarian working with Thoroughbreds.

I know many fine and forward-looking men in your field who are working to elevate standards and institute improvements. Through their efforts they have encouraged The Jockey Club to sponsor and support the project of blood typing and grouping. At the dinner of the Thoroughbred Club of America in October, I reported on this project, and although this may be somewhat repetitious to some of you who may have read my report, I think the project as a whole is of such great importance to the Thoroughbred industry that I should like to repeat the text to some extent.

Nearly a year ago The Jockey Club gave a grant to the Jewish Memorial Hospital to make a study of the Thoroughbred's blood under the supervision of a crew consisting of Doctors Alfred Schwarz, Harry Wallerstein and Manuel A. Gilman, who is The Jockey Club's veterinarian.

The project has reached a point where now we believe that without doubt the Thoroughbred's blood can be broken down into more types and groups than human blood. I think to veterinarians the greatest benefit from this project is the fact that it will enable horses to have transfusions with security, whereas, as you well know, the possibility of blood being incompatible has made transfusions a definite hazard. The inability to replace blood when needed has retarded progress in veterinary

surgery, particularly among the extremely valuable Thoroughbreds.

I look forward to tremendous progress in surgery in saving the lives and usefulness of many of our most valuable animals. Of great importance to the racing industry but of minor importance to the veterinary field is the achievement of determining parentage by way of negative tests. There is also the matter of identification of an animal as blood typing adds one more factor in proving or disproving identity.

In closing I would like to advise you to be tolerant of the problems that may seem important to you and your fellow equine practitioners on your first analysis, because it is impossible to legislate for and rule an industry that is as large as racing without all of us giving a little, even in the items that may seem specially aimed at us. As a racing official I felt for a long time that I was best equipped to write the rules which control the supervision of racing. I soon learned that I would naturally write a rule which made it easy for me to operate, rather than a rule which might be difficult to enforce but did promote the best interests of all divisions of racing. In fact, it was because of such experiences that The Jockey Club inaugurated the Round Table Conferences which provide a medium for all problems of racing to be discussed by every section of the industry before action by any single power or group. No longer is it possible for a proposal for action which may adversely affect other groups in racing to be passed before being considered by all.

The Jockey Club, the National Association of State Racing Commissioners, the Thoroughbred Racing Association of the United States and all Stewards who are held responsible for the integrity and welfare of racing are deeply conscious of the great value the science of veterinary medicine and surgery has been to racing. It has been my great pleasure and privilege to have worked with such men as Drs. James G. Catlett, Manuel Gilman, Jordan Woodcock and William Reed and all the very capable practitioners in New York and elsewhere. It has been a great source of satisfaction that Dr. Catlett and other veterinarians have temporarily at least accepted posts as Stewards at some of our most important racing centers. In all cases they have performed in a manner that brought great credit to the veterinary profession. That is particularly fine because they could bring to the official operation a knowledge that could only be obtained through training with the animal in a professional manner. They are truly horsemen and come by that title because they have learned what makes the Thoroughbred tick by way of hard study and training.

Thank you very much for having asked me here and for listening to my ideas.

## LOCAL ANESTHESIA

By A. H. DAVIDSON, D.V.M.

Lexington, Kentucky

Mr. Chairman-Fellow Veterinarians:

I was assigned the subject of "Local Anesthesia" for this meeting and the subject is a good one. The only difficulty is the fact that I know very little about it. I do use it in quite large quantities but with little thought or knowledge of what I am doing other than accomplishing an end.

During the fall and winter I spent most of my time firing horses. Consequently when I think of using local anesthesia it is with reference to firing.

### FRONT ANKLE

The difficult part of blocking an ankle for, for instance firing, is getting a start at some point. It seems easiest for me to pick the foot up and step around in front of the horse and lay the flexed knee across my lap. Usually a helper holds the foot, but not always. I then ease the small needle into the skin at an oblique angle to the skin forcing anesthesia into the skin as the needle slowly enters it. Inject from 1 to 2 cc subcutaneously at a spot and force the wheal ahead so that the next injection will be at the farthest edge of the wheal. By not hurrying too fast thus giving the anesthetic a chance, the horse may not feel any of the injections.

It seems to me the easiest starting point is directly over the superficial tendon. I then work both ways across both descending nerves and on around the leg above the fetlock until I meet over the front of the metacarpal. The front of the ankle will be the most sensitive and the last area to become anesthetized. After I have completed the ring around the leg I proceed with the other leg, presuming now that both ankles are to be fired. After the second leg is circled I sometimes go around each leg slightly distal to my first injections but using smaller doses of anesthesia. If I don't do this the front of the ankle and pasterns are slow to lose sensation.

It is now possible to start firing but I stay high for 5-10 minutes and when I do run the line of points on down to the pastern I do it lightly until I'm sure the leg is anesthetized.

I remove twitch when the first ring is completed and seldom have to apply it. Smoke will sometimes make trouble and the horse may have to be turned into the direction of the air currents, if any.

## KNEE

To block the knee I use the same procedure, starting over the extensors above the knee in front and working medially and laterally. Medially I cross above the epiphysis and then run straight down to the metacarpal. Laterally I cross the extensors and down to the metacarpal. It is not necessary to cross the lower part of the knee. The lateral surface above the knee over the superficial branch of ulnar is a very sensitive area and injections are small and close together.

I have never had much success blocking the knee relative to finding lameness. Most always a knee will tell you when it is sore by flexion and sometimes pressure on the extended leg applied at the knee.

## SPLINTS

Splints are infiltrated with anesthesia. Large splints I inject a ring around them and the small ones infiltrate over them.

## TENDONS

When a tendon is to be fired I inject under the knee at the very top of the tendon extending the infiltration out on the metacarpal both medially and laterally. However, down the leg at about where the anastomatic branch crosses the tendon another infiltration is placed extending out to or beyond the suspensory ligament. The lateral surface of the leg is readily anesthetized but the inside is treacherous and difficult to block. If I am in a hurry to get on with the firing, I use excessive doses of anesthesia. If I have two or more horses to fire in the same stable I will anesthetize two and then fire two. This gives the anesthesia a better chance and less can be used.

## PASTERNS

Pasterns are anesthetized by blocking the digital nerves and infiltrating across the front of the pastern.

## HOCKS

Hind legs require, sometimes, a certain amount of acrobatics depending on the horse. Seldom have I used nerve blocks as an aid to lameness diagnosis of the hind legs. However, it can be of use if the lameness is low. The use of local anesthesia on the hind leg is relative to surgery, most often, firing. I have always infiltrated the areas in the region of the hock which are to be fired. It is true that the hock can be anesthetized by blocking the nerves but I do not feel comfortable behind or under the horse if I use this method.

On curbs I infiltrate a ring around the area to be fired. The start is usually below the cap of the hock and lateral to the superficial flexor tendons. From there I go across to or beyond the night eye on the inside and down on the lateral aspect. I inject down over the head of the lateral splint bone and cross to inside. The area inside the tendon directly below the night eye is the one to watch as it is a very sensitive area.

In most cases a twitch and the front foot held up is enough restraint. Ethyl chloride may be useful on a touchy horse to get the injections started. On some horses it is necessary for an assistant to hold the hind foot up but it is awkward to get at the leg in this position.



## AN ABSTRACT

### Safety of Piperazine-Carbon Disulfide Complex in Foals, Weanlings, and Broodmares

NOLEN D. CONNOR, D.V.M., M.S.

The Upjohn Veterinary Experiment Station  
Richland, Michigan

In discussing the safety of piperazine-carbon disulfide complex in foals, weanlings and broodmares, I would like to present a resume of a number of experiments with a specially tailored formulation called Parvex Suspension®. Parvex is a polyvalent anthelmintic discovered by Boots, Ltd., of England, and intensely investigated by both Boots and The Upjohn Company. The compound is an equimolecular complex of piperazine and carbon disulfide which dissociates into piperazine and carbon disulfide in weak acid solutions, such as found in the stomach, providing two effective anthelmintic agents. Parvex Suspension was formulated to facilitate administration of the compound to horses by intubation.

In this paper the effect of Parvex Suspension on parasite egg counts and parasite elimination will not be considered. This will be discussed in papers presented later this afternoon. The safety of the preparation and the toxicological manifestations of overdosage are from the results of experiments involving animals at our experiment station over a period of two years.

An effective dose of 0.5 fluid ounce per 100 pounds of body weight has been established for the removal of ascarids and small strongyles. A dose of 1.0 fluid ounce per 100 pounds is more effective for the elimination of bots, pinworms, and large strongyles.

### CONCLUSIONS

1. Parvex Suspension has been administered to 141 foals, 91 weanlings, and 235 broodmares of various breeds in doses ranging from 1 to 12 times the recommended dose under a wide variety of conditions in every season of the year. Normal healthy animals did not show severe toxic reactions until 12 times the efficacious dose was given.
2. The treatment of barren mares with Parvex Suspension during the breeding season resulted in no untoward effects on the estrous cycle or the conception rate.

3. Pregnant mares treated up to 1 day prior to delivery had normal parturition and delivered normal healthy foals. Treatment produced no discernible adverse effects on gestation.
4. Foals as young as 2 days old whose dams were treated with Parvex Suspension have shown no ill effects from drinking the milk.
5. In one experiment with nursing foals, 47 foals administered up to 6 times the effective dose showed no toxic manifestations while 5 of 8 foals administered 12 ml. of carbon disulfide showed symptoms of carbon disulfide intoxication.
6. The administration of 12 times the effective dose to weanlings produced no deaths.
7. The administration of 8 times the recommended dose to a convalescing weanling in a weakened condition produced severe toxic manifestations, but healthy weanlings of the same age similarly treated showed no ill effects.
8. The administration of up to 12 times the recommended dose of Parvex Suspension to foals, weanlings, and broodmares has not resulted in any unfavorable changes in hemograms of the animals as denoted by changes in erythrocytes, hemoglobin, packed-cell volumes, leukocytes, or differential counts.
9. Heavily parasitized animals have always shown an increase in erythrocytes, hemoglobin, and packed-cell volumes following expulsion of the intestinal parasites after treatment with Parvex Suspension.
10. A suggested treatment for carbon disulfide intoxication and for overdosage with Parvex Suspension is the intravenous administration of Solu-Cortef®, 100 mg. and 10% glucose.
11. Parvex Suspension administered according to directions to horses is an exceptionally safe and effective anthelmintic.

# CRITICAL TESTS AND FIELD STUDIES WITH PARVEX\* AGAINST PARASITES OF THE HORSE\*\*

J. H. DRUDGE, D.V.M., and S. E. LELAND, JR., D.V.M.

Department of Animal Pathology  
Kentucky Agricultural Experiment Station  
Lexington, Kentucky

## INTRODUCTION

Piperazines have been recently introduced as anthelmintic agents in man and domestic animals. Several of these compounds are available commercially. The special qualities found in piperazines are the low order of toxicity and a range of activity against several types of roundworm parasites.

In the horse, most of the investigations have been on piperazine adipate, and the activity is characterized by (1) a high degree of efficiency against *Parascaris equorum* and small strongyles, (2) a 50-60 percent removal of *Strongylus vulgaris*, the causative agent of verminous aneurysm, and (3) an 80 percent removal of mature pinworms, *Oxyuris equi*.

At the outset, the piperazine-carbon disulfide complex appeared to offer special value in the horse where the action of the carbon disulfide component against bots and ascarids would be combined with the activity of the piperazine base against ascarids, strongyles and pinworms. Such polyvalent anthelmintic action has been hitherto unavailable in a single compound.

Our evaluation of this compound has been made in two phases; (1) critical tests and (2) field studies. Details of the critical tests and the 1956 field studies have been published elsewhere 1, 2. The purpose of this discussion is to summarize our findings, including this year's field studies which are still in progress.

## OBSERVATIONS

*Critical Tests* (Table 1): Eight animals were used in this phase of our investigations, and the dose of piperazine-carbon

\* Suspension of the piperazine carbon disulfide complex, 250 mg/cc (Upjohn Company).

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Table 1. Percentage of Efficacy of Parvex in the Horse as Determined by Critical Tests.

Horse	Dose		Bots		Ascarids (P.e.)		Pinworms (O.e.)		Strongyles	
	Rate mg/kg	Total cc	G.i.	G.n.	Mat.	Immat.	Mat.	Immat.	S.v.	Large S.ed. S.eq.
M 615	75	125	100	38	-	-	-	-	-	-
W 62	75	60	100	100	100	64	-	-	-	-
M 6L	100	225	17	92	-	-	-	-	-	-
Y 621	100	95	81	63	100	-	75	7	65	-
Y 622	150	150	47	100	100	100	-	16	34	2
Y 620	150	170	66	-	100	-	-	0	59	0
Y 623	200	235	51	100	100	-	-	28	39	38
Y 624	200	170	100	-	-	-	-	15	40	20
										0
										95

G.i. = *Gastrophilus intestinalis*  
 G.n. = *Gastrophilus nasalis*  
 P.e. = *Parascaris equorum*  
 O.e. = *Oxyuris equi*

S.v. = *Strongylus vulgaris*  
 S. ed. = *Strongylus edentatus*  
 S. eq. = *Strongylus equinus*

Table 2. 1956 Field Studies. Egg Count Data on Efficacy of Parvex Against Ascarids and Strongyles.

Animals			Post-Treatment (Week)											
Group	Age	No.	Pre-Treatment	1	3	4	6	8	10	12	13	15		
Ascarid Eggs Per Gram Feces - Group Average														
C 1	S	7	525	0	0	0	0	0	0	0	0	0	0	
E 1	W	8	310	0	0	0	0	0	0	0	0	0	225	
L 1	W	6	10	0	0	0	0	0	0	0	260	0	0	
L 2	W	14	165	0	0	0	0	0	15	0	80	0	0	
M 1*	W	12	0	0	0	0	0	0	0	0	0	0	150	
M 2*	W	10	0	0	0	0	0	0	0	0	0	0	0	
L 3	Y	6	260	0	0	0	0	0	0	35	0	0	0	
L 4	Y	14	80	0	0	0	0	0	65	0	0	0	0	
M 3*	Y	11	150	1	0	0	0	4	0	20	0	0	0	
M 4*	Y	10	145	0	0	0	0	0	0	235	0	0	0	
M 5*	Y	20	150	8	8	8	8	4	0	10	0	0	0	
E 2	Y	5	225	0	0	0	0	0	0	0	0	0	0	
W 1*	Y	16	55	0	0	0	0	0	0	0	0	0	0	
W 2*	Y	14	35	0	0	0	0	0	0	0	0	0	0	
M 6*	Y	10	20	4	0	0	0	0	0	0	0	0	0	
M 7*	Y	6	335	1	0	0	0	0	0	0	0	0	0	
E 3	M	4	0	0	0	0	0	0	0	0	0	0	0	
J 1	M	3	0	0	0	0	0	0	0	0	0	0	0	
Strongyle Eggs Per Gram Feces - Group Average														
C 1	S	7	545	15	15	25	110	220	0	0	0	0	0	
E 1	W	8	800	5	10	25	655	0	0	455	0	230	0	
L 1	W	6	230	5	0	0	0	0	0	0	1040	0	0	
L 2	W	14	340	10	0	0	1480	0	1360	0	1480	0	0	
M 1*	W	12	40	2	0	2	0	70	0	130	0	105	0	
M 2*	W	10	25	0	0	5	0	15	0	125	0	0	0	
L 3	Y	6	1040	45	0	0	340	0	320*	0	0	0	0	
L 4	Y	14	1480	50	0	5	400	0	195*	0	0	0	0	
M 3*	Y	11	105	5	0	5	0	130	0	270	0	0	0	
M 4*	Y	10	125	5	0	3	0	85	0	215	0	0	0	
M 5*	Y	20	265	10	0	8	0	55	0	115	0	0	0	
E 2	Y	5	230	10	0	0	0	235	0	0	0	0	0	
W 1*	Y	16	155	1	0	0	0	0	0	0	0	0	0	
W 2*	Y	14	135	1	0	0	0	0	0	0	0	0	0	
M 6*	Y	10	270	4	0	0	0	0	0	0	0	0	0	
M 7*	Y	6	215	4	0	0	0	0	0	0	0	0	0	
E 3	M	4	1165	160	95	195	210	585	0	1625	0	0	0	
J 1	M	3	1330	10	0	0	0	0	0	0	0	0	0	

+ = 18.75 gm. phenothiazine included in dose. \* = On low-level phenothiazine.

disulfide complex ranged from 75 to 200 mg/kg. The 75 mg/kg dose rate is equivalent to one-half ounce of the suspension per 100 pounds body weight, which delivers approximately 20 mg piperazine base per lb. and 4.5 drams of carbon disulfide per 1,000 lbs. body weight. Administration was via stomach tube, and the grain ration was withheld on the day of treatment.

The data on these critical tests are summarized in Table 1 by expressing for the various parasites the percentage that was removed by treatment.

Two species of bots were encountered and the removal activity was variable. For the common bot (*Gastrophilus intestinalis*), removal ranged from 17 to 100 percent, averaging 70 percent, and for the throat bot (*G. nasalis*) the range was 38 to 100 percent, averaging 82 percent. Increasing the dose rate did not necessarily enhance the activity.

Complete removal of ascarids (*Parascaris equorum*) was observed. Immature ascarids were present in two of the animals, the weanling (W62) harboring a total of 343 which were completely removed at the lowest dose rate administered.

Two animals were harboring adult pinworms (*Oxyuris equi*), and the observations credit the drug with removing 64 and 75 percent, respectively. Heavy infections of immature pinworms were present in all but one yearling but removal activity was variable and of a relatively low order.

Of the three species of large strongyles, *Strongylus vulgaris* was most effectively removed. The level of activity varied from 34 to 65 percent, and increasing the dose from 100 mg/kg to 200 mg/kg did not enhance the activity against this species. Only limited activity against *S. edentatus* was observed, and this came at the highest dose level, 200 mg/kg. A light infection of *S. equinus* was encountered in one animal, and no drug activity was found.

All strongyles other than the three *Strongylus* spp. are recorded as small strongyles in Table 1. A consistently high level of activity against these forms was observed, ranging from 89 to 98 percent.

*1956 Field Studies* (Table 2 and 3): All age groups on several farms in the central Kentucky region were included in the study. The treatment procedure consisted of withholding the grain ration on the morning of treatment, estimation of body weight, and administration of the suspension of piperazine-carbon disulfide complex via stomach tube. Dose rate was 0.5 ounce of the suspension per 100 lb. body weight, except to groups M6 and M7 which received a lower rate or .33 oz/100 lbs. The efficacy of treatment was judged from pre- and post-treat-

ment worm egg and larval counts on fecal samples. Only one pre-treatment count was made, whereas several post-treatment counts were made at intervals over periods as long as 15 weeks.

The eggs per gram (EPG) observations are presented as averages for each group in Table 2. A total of 176 animals were included in the study.

Only one of the 16 groups of young animals did not have ascarid eggs before treatment, and ascarid EPG's made one week after treatment were consistently negative except for 1 animal in each of groups M3, M6, and M7, and 3 animals in group M5. The reduction of ascarid EPG for these 6 animals averaged 91 percent. Of special interest was the failure of ascarid eggs to reappear in the feces until the tenth week after treatment.

Striking reduction of strongyle EPG was a characteristic finding in the examinations made one week after treatment. More complete removal of strongyle eggs was observed in yearling group W1 and W2 that were given 18.75 gm of phenothiazine along with the dose of piperazine-carbon disulfide complex. Tendency for strongyle EPG to increase was not noticed until the fifth week post-treatment. Generally, lower strongyle egg counts, before and after treatment, were associated with the low-level phenothiazine medication in groups with the "M" designation.

The larvae per gram (LPG) counts are presented as averages for each group (Table 3). The larvae of one large strongyle, *S. vulgaris*, were differentiated from all other strongyle larvae, most of which were small strongyles.

In contrast with the foregoing strongyle egg counts, the number of *S. vulgaris* larvae was not reduced by treatment. This was particularly evident in groups L3 and L4. Larvae of this species were not present in the pre-treatment cultures prepared from the weanlings and did not appear until several weeks later. Practically no *S. vulgaris* larvae were found in the animals that were on low-level phenothiazine treatment.

In general, striking reductions in other strongyle larvae followed treatment, corresponding with the drop in strongyle EPG. Furthermore, the number of these larvae did not increase until the fifth week, which is in accord with the egg counts. Comparatively small numbers of larvae were recovered from cultures prepared from animals that were on low-level phenothiazine treatment.

Several animals in groups M3 and M4 showed impairment of appetite and voided feces of softened, mushy consistency

Table 3. 1956 Field Studies. Larval Count Data on Efficacy of Parvex Against Strongyles.

TABLE 5. 1950 Field Studies. Larval Count Data on Efficacy of 1-Avex Against Strongyle

Animal

Post-Treatment (Week)

Group	Age	No.	Pre-Treatment	1	3	4	6	8	10	12	13	15
<i>S. vulgaris</i> larvae/gm. feces - group average												
L 1	W	3	0	0	-	-	5	-	-	-	7	-
L 2	W	5	0	0.1	-	-	-	17	17	-	23	-
M 1*	W	12	0	0	-	0	-	0	-	0.1	-	0
M 2*	W	10	0	0	0	0	-	0	-	0	-	-
L 3	Y	3	7	18	-	-	28	-	0.0*	-	-	-
L 4	Y	8	23	28	-	-	37	-	0.2*	-	-	-
M 3*	Y	11	0	0	-	0	-	0	-	-	-	-
M 4*	Y	10	0	0	-	0	-	0	-	-	-	-
E 2	Y	3	3	2	3	-	-	6	-	-	-	-
M 6*	Y	10	0	0	-	-	-	-	-	-	-	-
M 7*	Y	7	0	0	-	-	-	-	-	-	-	-
J 1	M	3	0	0.3	-	-	-	-	-	-	-	-

Other Strongyle larvae/gm. feces - group average

L 1	W	3	180	1	-	-	125	-	-	-	94	-
L 2	W	5	200	2	-	-	-	205	326	-	252	-
M 1*	W	12	2	0	-	1	-	10	-	24	-	32
M 2*	W	10	4	0	0.2	0.1	-	5	-	18	-	-
L 3	Y	3	94	12	-	-	65	-	3*	-	-	-
L 4	Y	8	252	14	-	-	74	-	26*	-	-	-
M 3*	Y	11	32	1	-	4	-	38	-	-	-	-
M 4*	Y	10	18	0	-	1	-	18	-	-	-	-
E 2	Y	3	175	19	2	-	-	42	-	-	-	-
M 6*	Y	10	29	2	-	-	-	-	-	-	-	-
M 7*	Y	7	37	1	-	-	-	-	-	-	-	-
J 1	M	3	380	4	-	-	-	-	-	-	-	-

\* = On low-level phenothiazine.

during the day following treatment, the only untoward effects ascribable to the treatment.

*1957 Field Studies* (Tables 4, 5 and 6): These trials designed for a direct comparison of Parvex and carbon disulfide with special interest in the control of ascarid infection. Two farms are included in this report, and the observations were started in the animals as sucklings, and are to be continued until they are yearlings. Both drugs were administered via stomach tube.

Efficacy of treatment was based on egg counts (EPG) which were made at two-week intervals. Changes in EPG immediately following treatment are used as an index of removal of mature worms, whereas subsequent counts serve as an index of activity against the immature ascarids.

*Farm L.* The foals on this farm were divided into two groups, one to be treated with Parvex and the other with carbon disulfide. The treatments and ascarid EPG's are summarized in Table 4.

At the time of the first treatment on 11 June, the ages of the foals ranged from 8 to 12 weeks and the estimated weights from 300 to 500 lb. Parvex was given at the rate of 0.5 oz/100 lb, and the total doses varied from 1.5 to 2.5 oz. In the carbon disulfide group the doses varied from 8 to 12 cc. The morning grain ration was withheld on the day of treatment.

This Parvex treatment was completely effective in eliminating the ascarid eggs from the feces, and none were detected until the 9th week post-treatment or after the prepatent period had elapsed. In contrast, the first carbon disulfide treatment failed to change the ascarid EPG in one of two foals showing ascarid eggs before treatment, and three of the animals, in addition to No. 8, became positive on the sixth week, which indicated incomplete removal of the immature worms. The animal that failed to respond was given another treatment of 12 cc of carbon disulfide on 8 July which was only partially effective. By the ninth week after the first treatment, all of the carbon-disulfide-treated animals were showing ascarid eggs, and the average count was much higher than the corresponding Parvex-treated animals.

The second general treatment was administered on 13 August when the estimated body weight range was 400 to 600 lb. The doses of Parvex suspension ranged from 2 to 3 oz, and of carbon disulfide from 15 to 18 cc. Morning grain ration was withheld on the day of treatment.

Again Parvex treatment appeared to be completely effective against both mature and immature ascarids. Ascarid eggs

Table 4. 1957 Field Study Comparing Parvez with Carbon Disulfide for Ascarid Control in Sucklings and Weanlings, Farm L.

Foal Number	Ascarid EPG and Treatment on Date Indicated															
	6/11		6/18		7/8		7/23		8/13		8/27		9/10		9/24	
	A	T	A	T	A	T	A	T	A	T	A	T	A	T	A	T
1	0	P	0	-	0	-	0	-	60	P	0	-	0	-	0	-
2	0	P	0	-	0	-	0	-	70	P	0	-	0	-	0	-
3	70	P	0	-	0	-	0	-	20	P	0	-	0	-	0	-
4	0	P	0	-	0	-	-	-	100	P	-	-	0	-	0	-
5	0	P	0	-	0	-	0	-	0	P	0	-	0	-	0	-
6	2310	P	0	-	0	-	0	-	0	P	0	-	0	-	790	-
7	0	P	0	-	0	-	0	-	80	P	0	-	0	-	0	-
8	6730	C	6060	-	14050	C	3830	-	3460	C	0	-	0	-	0	-
9	0	C	0	-	0	-	20	-	180	C	20	-	40	-	110	-
10	0	-	-	-	430	C	-	-	830	C	20	-	20	-	160	-
11	0	C	0	-	0	-	0	-	90	C	-	-	0	-	10	-
12	50	C	0	-	0	-	0	-	440	C	0	-	0	-	50	-
13	0	C	0	-	0	-	50	-	1090	C	30	-	80	-	120	-
14	0	C	0	-	0	-	10	-	650	C	-	-	260	-	30	-
															440	-

A = Ascarid eggs/gm. feces (EPG)

T = Treatment

P = Parvez, Suspension,  $\frac{1}{2}$  oz/100 lbs. body weight.

C = Carbon disulfide, 2.5-3.5 cc/100 lbs. body weight.

reappeared in two of the animals ten weeks after the second treatment. The 13 August carbon disulfide treatment markedly reduced the ascarid EPG's, but four animals retained low-grade infections of mature worms following treatment and during the ensuing between-treatment period. By the 10th week, six of the seven foals were ascarid egg positive.

The third general treatment date was 5 November when the estimated body weights ranged from 500 to 700 lbs. Doses of Parvex suspension were 1.75 to 2 oz. for the P1 group, and from 5 to 6 drams. Both drugs were completely effective in reducing the EPG's to zero. One observation date in addition to the last on the chart likewise had only negative counts.

*Farm N.* The animals on this farm fell into two groups according to age, each of which was divided into three treatment groups, viz., (P1) Parvex suspension .5 oz/100 lb, (P2) Parvex suspension .25 oz/100 lb, and (C) carbon disulfide.

The treatments and egg counts on the older age group are summarized in Table 5. At the time of first treatment of these animals on 3 July, the ages varied from 12 to 18 weeks, and the estimated body weights ranged from 350 to 425 lb. Doses of Parvex suspension were 1.75 to 2 oz for the P1 group, and .75 to 1 oz for the P2 group, while 4 to 5 dram doses of carbon disulfide were given to the C group. The morning grain ration was withheld on the day of treatment for the foals receiving the Parvex suspension. In addition to this, the carbon-disulfide-treated foals were muzzled for 3 hours before and 2 hours after treatment.

The P1 (0.5 oz/100 lb) dose of Parvex was completely effective in the two animals showing pre-treatment ascarid egg counts, and no eggs were detected until the 10th week, except for an EPG of 10 in No. 4 on the 8th week.

The P2 (.25 oz/100 lbs) dose of Parvex reduced the EPG to zero in 3 of 4 animals, and by 67 percent in the fourth animal. The latter animal spontaneously eliminated its ascarid infection between the second and fourth weeks. Eggs reappeared in No. 10 on the fourth week and spontaneously disappeared after the 8th week. By the 10th week, two of the P2 group animals were again showing ascarid eggs.

Two of the carbon-disulfide-treated animals had positive EPG's before first treatment. The count of No. 17 was reduced to zero, but the infection in No. 21 was refractory to treatment. A third animal (No. 18) in this group became positive on the second week after treatment which indicated incomplete removal of immature forms. The refractory animal experienced a spontaneous reduction during the 4 to 6-week in-

Table 5. Field Study Comparing Parvex with Carbon Disulfide for Ascarid Control in Sucklings and Weanlings. Farm N.

Foal Number	Ascarid EPQ and Treatment on Date Indicated																							
	7/3		7/17		7/31		8/14		8/28		9/11		9/25		10/9		10/23		11/7		11/20			
	A	T	A	T	A	T	A	T	A	T	A	T	A	T	A	T	A	T	A	T	A	T		
1	510	P1	0	-	0	-	0	-	0	-	0	P1	0	-	0	-	0	-	0	-	70	-		
2	0	P1	0	-	0	-	0	-	0	-	170	P1	0	-	0	-	0	-	0	-	0	-		
3	0	P1	0	-	0	-	0	-	0	-	60	P1	0	-	0	-	0	-	0	-	170	-		
4	0	P1	0	-	0	-	0	-	10	-	0	P1	0	-	0	-	0	-	0	-	0	-		
5	160	P1	0	-	0	-	0	-	0	-	100	P1	0	-	0	-	0	-	0	-	20	-		
9	750	P2	230	-	0	-	0	-	0	-	0	P2	0	-	0	-	0	-	0	-	0	-		
10	210	P2	0	-	30	-	90	-	70	-	0	P2	0	-	0	-	0	-	0	-	0	-		
11	2360	P2	0	-	0	-	0	-	0	-	20	P2	110	-	30	-	0	-	0	-	0	-		
12	1270	P2	0	-	0	-	0	-	0	-	110	P2	0	-	0	-	0	-	0	-	0	-		
17	1460	C	0	-	0	-	0	-	0	-	0	C	0	-	0	-	0	-	0	-	0	-		
18	0	C	40	-	70	-	40	-	0	-	0	C	0	-	0	-	0	-	70	-	0	-		
19	0	C	0	-	0	-	0	-	0	-	0	C	0	-	0	-	0	-	120	-	1120	-		
21	880	C	1100	-	1780	-	10	-	0	-	0	C	0	-	0	-	0	-	50	-	130	-		

A = Ascarid eggs/gm. feces (EPQ)

T = Treatment

P1 = Parvex suspension,  $\frac{1}{4}$  ounce/100 lbs. body weight.

P2 = Parvex suspension,  $\frac{1}{4}$  ounce/100 lbs. body weight.

C = CS<sub>2</sub>, 1 dram/month of age, maximum dose = 5 drams.

terval. A second carbon disulfide treatment was given on the sixth week after the first. Each animal received 5 drams and the same fasting procedure as before was followed. This treatment removed the residual infection in the two foals and there was no reappearance during the ensuing month.

The second Parvex treatment and the third carbon disulfide treatment were given on 11 September. The P1 animals weighed from 350 to 425 lb and received 1.75 to 2:15 oz of Parvex suspension, and the P2 animals weighed 300 to 400 lb and received .75 to 1 oz of the suspension. All of the carbon-disulfide treated foals received 5 drams. The same fasting procedures were followed.

Both Parvex dose levels were completely effective except in animal No. 11 which was in the P2 group. This infection was refractory to treatment, but spontaneously disappeared between the 4 to 6 week post-treatment interval. Ascarid eggs reappeared in three of the P1 animals on the tenth week.

None of the four carbon-disulfide-treated animals had ascarid EPG's at the time of the third treatment, but three became positive on the 8th week after treatment.

The younger age group of foals on Farm N were given their first treatment on 14 August (Table 6). Their ages varied from 12 to 16 weeks and their estimated weights ranged from 300 to 400 lb. Total doses of Parvex suspension were 1.5 to 2 oz for the P1 foals and .75 to .9 oz for the P2 foals. Doses of carbon disulfide were 4 to 5 drams. The same fasting procedures indicated for the older age group were followed on these younger animals.

Only 1 of the P1 foals had an ascarid egg count before treatment and all three were negative during the 8-week post-treatment interval. Two of the P2 foals showed eggs before treatment and both were negative two weeks post-treatment. However, ascarid eggs reappeared in three of the four P2 foals by the 8th week.

Only 1 of the carbon-disulfide-treated foals had a pre-treatment egg count and treatment reduced the EPG by about one-third. In addition to the incomplete removal of the mature worms in the foregoing foal, incomplete removal of immature worms from Nos 22 and 24 was indicated by the appearance of ascarid eggs on the fourth week.

The second carbon disulfide treatment was given four weeks after the first, and each foal received 5 drams. This treatment was completely effective as ascarid eggs disappeared from all three positive animals and no eggs were found during the post-treatment 4-week interval.

Table 6. 1957 Field Study Comparing Parvex with CB, for Ascarid Control in Sucklings and Weanlings. Farm N.

Foal Number	8/14		8/28		9/11		9/25		10/9		10/23		11/6		11/20	
	0		2		4		6		8		10		12		14	
	A	T	A	T	A	T	A	T	A	T	A	T	A	T	A	T
6	0	P1	0	-	0	-	0	-	0	P1	0	-	0	-	0	-
7	0	P1	0	-	0	-	0	-	0	P1	0	-	30	-	30	-
8	120	P1	0	-	0	-	0	-	0	P1	0	-	0	-	0	-
13	0	P2	0	-	0	-	0	-	290	P2	0	-	0	-	0	-
14	0	P2	0	-	0	-	0	-	0	P2	0	-	0	-	0	-
15	40	P2	0	-	0	-	200	-	10	P2	0	-	0	-	0	-
16	100	P2	0	-	10	-	140	-	20	P2	0	-	0	-	0	-
20	1530	C	1010	-	1220	C	0	-	0	C	0	-	0	-	0	-
22	0	C	0	-	130	C	0	-	0	C	0	-	-	-	-	-
23	0	C	0	-	0	C	0	-	0	C	0	-	0	-	0	-
24	0	C	0	-	70	C	0	-	0	C	0	-	0	-	0	-

A = Ascarid eggs/gm. feces (EPG).

T = Treatment.

P2 = Parvex Suspension,  $\frac{1}{4}$  oz/100 lbs body weight.  
C = CS2, 1 dram/month of age, maximum dose =  $\frac{5}{5}$  drams.P1 = Parvex Suspension,  $\frac{1}{2}$  oz/100 lbs body weight.

The second Parvex treatment and the third carbon disulfide treatment were given on 9 October. The P1 animals weighed 350 to 425 lb and received 1.75 to 2.15 oz of Parvex suspension, and the P2 animals weighed 300 to 350 lbs and recieved .75 to .9 oz of the suspension. Each of the carbon-disulfide-treated foals received 5 drams.

Both dose levels of Parvex appeared to be completely effective except for the P1 dose in foal No. 7. A low grade count was recorded on this animal on the fourth week after treatment which indicates the failure to remove some of the immature worms.

None of the carbon disulfide foals had patent ascarid infections before the third treatment, and no positive counts were observed during the six-week post-treatment period.

### SUMMARY

The critical tests demonstrated effective action of Parvex against several of the common parasites of horses. Complete removal of both mature and immature ascarids was found at dosages as low as 75 mg/kg (0.5 oz suspension/100 lbs). Removal activity against bots was variable but averaged 70 percent for *G. intestinalis* and 82 percent for *G. nasalis* following dosages ranging from 75 mg/kg to 200 mg/kg. Small strongyles were effectively removed (89-98 percent) by dosages varying from 100 mg/kg to 200 mg/kg. The latter dosages were less effective (34 to 65 percent) against *Strongylus vulgaris*, and much less effective (0-38 percent against *S. edentatus*. Activity against immature pinworms was of a low order (0 to 28 percent), while more complete (64 to 75 per cent) action on the adult forms was observed.

The 1956 field studies indicated the effective removal of both mature and immature ascarids and small strongyles from treatment with Parvex suspension at the rate of 0.5 oz/100 lb. This dose level showed no apparent action against *Strongylus vulgaris*.

The 1957 field studies comparing Parvex and carbon disulfide for ascarid control clearly demonstrated the superiority of Parvex suspension in dosages of 0.5 oz/100 lb. One-half this dose level (0.25 oz/100 lb) of the suspension was too low for effective control, but compared favorably with carbon disulfide under the conditions of these trials. Shortcomings of carbon disulfide which were evident included (1) incomplete action in removing mature ascarids, (2) no apparent action at all in an occasional animal, and (3) incomplete removal of immature ascarids.

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# THE USE OF PARVEX ON THOROUGHBRED FARMS

*By*

D. L. PROCTOR, JR., D.V.M.

Over a period of the last two and a half years I have had a supply of Parvex put at my disposal. The drug has always been in a suspension form and the dosage recommended has been 75 mgs. per kilo or what was referred to here as 1 oz. per 200 lbs. live weight. That has been the only dosage as I have personally used it, and the only form in which I have used Parvex.

I have used it on about 500 horses. I haven't had the facilities to conduct the egg per gram counts that Dr. Drudge has shown here on the slides, but from time to time we have checked groups of these animals on which we have used the Parvex and the fecal examinations have compared favorably with what my clients are used to. I am not a demon parasitologist. I'm perfectly willing to leave a few worms in my animals. I'm not at all convinced that it is absolutely essential to have a perfectly 0-0 count as long as these animals do not manifest any clinical symptoms, and as long as the post-treatment fecal counts are down below 200 or so, I feel that I have accomplished my purpose.

My routine procedure is starting at this time of the year. We all realize that the bot fly ceases to fly after the first hard frost, but many of us don't realize that these eggs that they have been depositing all through the months will hatch out according to the licking of the animal of the bot nits on their legs; and depending on friction and moisture that animal may have a continuous infection of bots for the next 6 to 8 weeks. Therefore I would seriously recommend your consideration of advising your clients that immediately after the first hard frost, (those of us who are fortunate enough to live up here where we do get a hard frost occasionally), a thorough washing with good hot water will cause those nits to embryonate at which time they are very susceptible to equal parts of kerosene and mineral oil just smeared on very lightly with a wet rag. That is a common sense, easy preventative measure in parasite control that many of us are prone to forget. As we discussed or mentioned yesterday, prevention is much better than treatment. That is one real good way to break the life cycle of the bot fly.

We ordinarily worm both barren and pregnant mares some time in the last part of Decmeber or the first part of January.

I just arbitrarily have adopted a program of my own of never worming any mare that is going to foal before the first of March. It is not that I do not think they can't be wormed, but I don't want to be responsible for any of the acts of God and nature that may take place after I've wormed one. And so, any of those mares that foal in January and February we arbitrarily decide not to worm for bots or ascarids in December or January. We worm them during the first 9 days post partem. And so far that has worked out very nicely. Now the later-foaling mares and barren mares we routinely worm in the last part of December or the first part of January.

Getting on with this worming program that we have been trying to outline, the foals are routinely wormed with Parvex or Piperazine as the case may be at 8, 12 and 16 weeks. Those three wormings seem to insure us of a relatively, ascarid free group of foals. Then relying on fecal examination as to when we worm again, the final worming usually doesn't take place till a suckling is weaned. Some people feel it is a bad time to worm because the foals are all upset. Others feel it is a darned good time to worm because they are already upset; you just upset them a little more, and it doesn't make any difference anyhow. I haven't any very pronounced feelings on the subject either way.

I think that most of us don't utilize the efficiency of a fecal examination the way we should. I'm old enough in the profession or have been associated with it long enough that I remember the days when we used to worm horses with tetrachlorethylene or carbon tetrachloride, and glauber salts for the babies for strongyles; and then followed that up, when they got old enough to stand the treatment, with oil of chenopodium, turpentine and linseed oil. In those days you never had to ask a man whether a group of stock had been wormed. 1) You could smell it from a distance of 100 feet from the barn. 2) You could tell the exact height of the anus by the splatter of the feces on the walls of the stall. And it was always a question as to which was worse, the worm medicine or the worms. I think that having been subject to that in my youth, I have a firm belief that horses are maybe like dogs and a few worms kind of keep them happy. Just like a few fleas keep a dog from getting psychotic.

We try to utilize fecal examinations in our worming procedure in yearlings and in older horses, and as long as infestations are relatively low, (if we have the facilities for e.p.g.'s (eggs per gram) anything below 400 will probably pass and anything below 1-x on this arbitrary system of x's), I'm not at all adamant about suggesting that they be wormed immediately. If you do have one horse that flares up very high

I think it is common sense to go in there and worm that particular animal, or any animal that's on the border line. But I do not recommend indiscriminate worming without proper fecal examinations just because you arbitrarily set up a program. In other words the program is fine; every farm must have it, but you can administer it with common sense.

Getting back again to Parvex as such, it is a nice preparation. It is well tolerated by the animals. It is of special use to me when I am away from home. I think it is effective. It is nice to worm horses and know that they are not going through this belly ache routine that so often accompanies administration of carbon disulphide. I was telling some of the gentlemen here about going out the other day and worming a group of Shetland ponies. We ended up the administration of the vermifuge with the gentlemen owner walking in with two fifty-pound ponies, one under each arm. Believe me, I didn't even have any faith in my graduates. I got out a 10 cc syringe, and started measuring out the vermifuge. My combinations are Parvex alone, Parvex and Phenothiazine in conjunction, Carbon Disulphide alone, Carbon Disulphide and Phenothiazine mixture, and Piperazine and Phenothiazine in mixture. And I use the Parvex where I feel that I've got animals who have a bot problem. If bots are not a problem, I switch over to Piperazine and if I feel that I've got a strongyle problem, I don't believe you destroy the efficiency of either drug if you mix the Piperazine and the Phenothiazine together. I do think that this new preparation does have a place in veterinary medicine in animals that are a little bit under par, valuable animals in racing condition, mares that are about to foal, and especially young animals; or where you are in a position where you are away from the scene of operations and you administer the vermifuge and then travel 50 miles. It is disconcerting to have a man call you up and say, "This mare is very colicky," and you have to make that 100 mile round trip to treat your intoxication.

That is about the sum and substance of my personal experience with Parvex over the last two and a half years.



## CLINICAL OBSERVATIONS WITH THE USE OF PARVEX IN STANDARDBREDS

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It is difficult to follow the distinguished men who have just preceded me in giving you a resume of their experiences with the use of Parvex. Everything that is necessary for you men to know has already been covered adequately. Therefore, I will only briefly relate some of my experiences with the use of the drug.

In order for you to better understand some of my statements, I should first explain that my practice is primarily with standardbred horses here in the Chicago area. I do not keep case records and, therefore, have no statistical records to which I can refer. I am only relating my observations as a practitioner.

In September 1957, Dr. Gordon Stocking contacted me through Dr. Foley, a good friend of mine. Dr. Foley was already using Parvex and recommended it to me. Dr. Stocking gave me about 30 doses of Parvex to be used experimentally on suitable clinical cases of parasitism. In the course of the next two and one-half months, I used Parvex on 19 horses which I considered good clinical cases. On this group of horses I ran fecals before worming and one week following worming, and on the initial fecal examination, all of the horses had egg counts of 2 plus to 4 plus.

I might explain here that at Michigan State College I was taught to classify egg concentrations found in fecal examinations in the following manner: The term one-plus designates the case where up to ten parasitic ova are counted in one low power microscopic field. Two-plus cases have 10 to 15 ova per field. Three-plus cases have 15 to 20 ova per field and four-plus cases have over 20 ova per field.

As previously stated, all of the 19 horses wormed had counts of two-plus to four-plus made up of ascarids, strongyles and an occasional oxyuris or pin worm. One week following worming, when the fecals were checked again, the four-plus cases had dropped to one-plus or two-plus, and the lighter cases, say two-plus, were generally negative. I considered these results good and I was sold on the product.

I think we all agree that race track practice is quite different from a farm practice and the relationship between the veterinarian and the trainers is quite important. I have always found that giving a horse a capsule for bolus with my hand creates a favorable impression with the trainer, especially those

trainers who have had personal experience with bleeders following the use of the stomach tube. My father taught me how to capsule a horse with my hand many years ago and I was further coached by Dr. Davidson when I spent a few weeks in Kentucky with the firm of Hagyard, Magee and Davidson. I know this is a controversial subject, but I have never been hurt. Once in awhile I get my forefinger raked, but I have eliminated that by using a little rubber gauntlet glove. When going through a stable of 15 to 20 horses I keep 3 or 4 such gloves in a bucket of novalsan solution and use a different glove for each horse.

Therefore, I contacted Dr. Stocking and asked about the possibilities of having Parvex put up in a bolus, so that I could administer the drug in that fashion and eliminate the use of the stomach tube. I must admit that I do not favor the use of a stomach tube where I can eliminate it. Just an occasional bleeder, especially where the blood is well distributed on a wall, does much to destroy your relationship with an owner or trainer. Also I am certain that I can save time worming horses by using a bolus. If I don't run into too many bad-acting horses, I dare say I can worm 40 horses with a bolus in the same time it takes to worm 15 with a stomach tube. I don't believe in doing anything the hard way if there is an easy way. Actually, the bolus suits me much better than using the stomach tube.

It took the Upjohn Company and Dr. Stocking some months to get a bolus in production and eventually they arrived in July. The first boluses produced were a 7.5 gram bolus which was the largest that the Upjohn Company could produce with their machinery. This meant that a thousand pound horse should receive about 4 boluses. This was very impractical if not impossible with some horses. Eventually a 20 gram bolus was produced.

In late July I started using the boluses to worm horses and I selected 29 horses as good clinical cases upon which to evaluate Parvex. All of these horses, as before, had egg counts of two-plus to four-plus. After worming, the counts were either negative or appreciably reduced. It is very gratifying to me to have an owner or trainer come around and tell me how well a horse is doing after being wormed, and most of them are greatly impressed with Parvex. Since Parvex is particularly effective against the round worms, the results are nothing short of spectacular to the layman who witnesses the passage of a large volume of these worms in the feces.

Including the 29 horses referred to previously, I have wormed 128 horses since last July. Only on those 29 horses were fecal checks made before and after worming, but clinically speaking, I feel that I have had uniformly good result on the other horses as well.

## PRACTICE TIPS CONTRIBUTED BY MEMBERS PRESENT AT THE 1957 MEETING

To reduce wrist fatigue encountered in dental work, weld the handle of a hacksaw to the shanks of commercial dental floats.

*Parascaris equorum* infestation of the horse is revealed upon fecal examinations by two different eggs.—One is brown covered; the other is thin walled with a dumbbell shaped nucleus. The latter is an immature egg and lacks albuminous covering.

The use of a twitch on sucklings and weanlings for the introduction of a stomach tube tends to constrict the nostrils and interfere with passage of the tube. Substitute tailing with manual pinching of medial margins of both nares with the hand holding the tube at the nostrils if the animal resists. This action has an effect similar to twitching and does not cause the foal to avoid being caught.

Clearly mark your stomach tube so that the mark is about four inches from the external nares just as the animal swallows. Exact knowledge of the whereabouts of the end of the stomach tube aids in getting the animal to swallow the tube and helps prevent passage into the trachea.

In pregnancy examination for the beginner, the introduction of a speculum into the vagina greatly facilitates the location and palpation of the uterus.

Stockinette (3 inches) makes a very suitable dressing for all leg and foot wounds and for use after firing. It is more rigid but gives to the bending of the limbs more readily than gauze.

"Windsucker" mares have conceived when treated with broad spectrum antibiotics, bred and sutured the same day. This treatment may also be of value on "repeater" mares many of which probably have a low grade endometritis and will show some non-hemolytic staph and coli when cultured while in heat. These mares are frequently reported "negative for significant bacteria".

Where horses must be destroyed at the race track a human anesthetic called Anectine (400-600 mg I. V.) may be used. The action is fast (5-10 seconds) and the horse does not struggle. This may be followed with Lethol or similar products to stop the heart.

Pentobarbital sodium and mephenesin (Myotal-Warren-Teed) is useful as a muscle relaxant in the treatment of colic and as a sedative for minor surgery with local anesthesia. Dose: 10 to 20 cc I. V.

Tincture of catechu in capsules helps control diarrheas that may follow influenza complications.

Thiamine Hydrochloride may be of value in the treatment of obscure mixed lamenesses.

For immunization against tetanus the use of the human packaged tetanus toxoid (Dose 0.5 cc to 0.75 cc) eliminates the swelling and extreme tissue reactions frequently seen with use of the veterinary product.

The use of a tranquilizer, following or at the time of firing, greatly reduces the discomfort to the horse and possible self-inflicted injuries that may result when the anesthetic wears off.

Succinyl choline chloride (20 mg. per cc) has been administered experimentally at the rate of 2 cc five times daily for fifteen consecutive days to several horses with no untoward side effects.

For the relief of pain in colic cases 15 cc of demerol hydrochloride (50 mg. per cc) is very useful in conjunction with the use of oils, etc. for correcting the bowel.

Violent colic cases respond nicely to the use of 5 cc of Sparine (Wyeth) and 10-15 cc of Novin (Haver-Lockhart) intravenously. Smooth as well as skeletal muscle relaxation results along with mental relief. Enemas, oils, antiferments, etc. as needed, are supplemental to this treatment.

The use of intravenous antihistamine immediately upon the discovery of any febrile type systemic disease seems to be of considerable help in checking the progress of the disease and hastening the recovery.

Hypodermic syringes and needles may be conveniently sterilized by using two stainless steel pans with covers. Each pan has a removable tray which rests on legs within the pan and has multiple holes throughout the bottom. The tray also has a raised section which is drilled with many small holes to hold various sizes of needles. Each tray may hold twelve 10 cc syringes and 20-30 needles. Sterilization may be accomplished by placing a layer of 2 cm. of water in the pan and boiling for 15 minutes. Pans may be alternated daily. Such a procedure will greatly reduce post-treatment abscesses and guard against carrying diseases from one animal to another.

As an aid in suturing wounds of the eyelids, nose and face of horses, wet the fingers with clean warm water. Take one or

two procaine tablets and place them in the wound. The moisture on your fingers will soften the tablets. Administer more procaine solution by inserting the needle through the wound to inject. After the sutures are in place and tied a few drops of udder infusion ointment such as used to treat mastitis in cattle may be applied above the wound so that it will run down around the sutures. Apply two or three times daily.

Racing blinkers are helpful in protecting wounds of the eyelids.

Salivary calculi should be removed near the opening of the duct at the level of the third or fourth upper check tooth. Use 3 to 4 cc of secostrin, mouth speculum and a hook knife. Always open within the mouth to prevent a salivary fistula.

The incidence of suspensory ligament lameness can be greatly reduced by keeping the toe as short as practicable and "dubbed off", with resultant raising of the heel.

